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ACOUSTIC TRAUMA IN MAN

CLINICAL AND EXPERIMENTAL STUDIES

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CHICAGO

Audiometric studies made during the past decade by Bunch,[1] Larsen,[2] Dickson and his associates,[3] McCord and his associates[4] and others have helped to crystallize in otologic practice the clinical entity of acoustic trauma. Extensive bibliographies on the subject appear in the papers of Bunch, Larsen, and McCord and his associates. Many types of sound stimuli are now known to be injurious to the ear, e. g., the noise of pneumatically driven tools, such as those used in riveting, chipping steel and breaking concrete; the noise of motors—gasoline (airplane), oil (Diesel) and electric; the noise of steam engines and whistles (locomotives); the noise of discharging firearms (large and small); single explosive sounds, such as that from a firecracker and that from a gas explosion, and the noise of machines, such as those used for grinding metal and for stamping out metal parts and drop hammers. A shock from a blow on the ear or a telephone receiver shock is also injurious.

When a group of employees in a noisy industry is examined, diagnosis is relatively simple, but when a large number of patients with mixed otolaryngologic signs and symptoms are cared for, the diagnosis is usually made by elimination. In a healthy person under 50 without history or evidence of aural disease and whose history does not suggest injury by intoxication from drugs, etc., or some severe constitutional

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1. Bunch, C. C.: Symposium: Neural Mechanism of Hearing; "Nerve Deafness" of Known Pathology or Etiology; Diagnosis of Occupational or Traumatic Deafness; Historical and Audiometric Study, Laryngoscope **47**:615-691 (Sept.) 1937.

2. Larsen, B.: Investigation of Professional Deafness in Shipyard and Machine Factory Labourers, Acta oto-laryng., 1939, supp. 36, pp. 3-255.

3. Dickson, E. D. D.; Ewing, A. W. G., and Littler, T. S.: Effect of Aeroplane Noise on Auditory Acuity of Aviators: Some Preliminary Remarks, J. Laryng. & Otol. **54**:531-548 (Sept.) 1939.

4. McCord, C.; Teal, E. E., and Witheridge, W.: Noise and Its Effect on Human Beings, J. A. M. A. **110**:1553-1560 (May 7) 1938.

disease or deafness in the family but reveals exposure to acoustic stimuli of a nature resembling those mentioned, a high tone loss must be suspected to represent the effect of acoustic trauma. This loss and the resultant impairment of hearing, if any, noted by the patient are not progressive if the patient ceases to be exposed to the stimulus. Tinnitus, which often accompanies impairment in hearing, varies as to duration and intensity and may endure after the acoustic insult. During the active stage of prolonged exposure, the auditory threshold may fluctuate, depending on whether the examination is carried out immediately after exposure (when impairment is more marked) or after a period of rest (when definite improvement can be demonstrated). It is now generally accepted that the first evidence of acoustic trauma often appears as a localized depression in the hearing around 4096 cycles, both for air and for bone conduction, with retention of good function for frequencies below and above this level. The drop in threshold may be restricted to this frequency even after many years of exposure, and, although it may spread to involve higher frequencies (for instance, with further injury), the subject usually is unaware of any hearing impairment at this stage and hence is not seen by an otologist at the time. As a consequence, this early phase of acoustic trauma escapes recognition for many years. As further trauma occurs, the threshold curve shows a depression for frequencies below 4000 cycles, and the hearing impairment for speech, especially for the whispered voice, becomes noticeable. When the audiometer was first introduced, the occasional appearance of a localized depression at 4096 cycles or of an abrupt high tone loss in large numbers of audiograms of apparently normal subjects directed attention to this type of threshold curve and soon led to observations strongly suggesting that at least one important etiologic factor was acoustic trauma. Instances of acoustic trauma are often forgotten by patients or are not considered important enough to mention. Thus, the lack of a history carefully elicited with the question of acoustic trauma in mind probably explains the fact that this diagnosis is still infrequently made by otologists.

Some of the factors which influence the degree of hearing loss from acoustic trauma are:

1. The total time of exposure. Often exposure for years results in only a slight localized drop in the threshold curve, without noticeable impairment of hearing.

2. The length of exposure in each period. The ear can recover with rest. Lack of rest periods is conducive to greater trauma.

3. The loudness of the sound stimulus. It is likely that only loud sounds cause damage to the ear.

4. The age of the subject. Older persons are probably more susceptible, and their auditory apparatus has less recuperative power than that of younger persons.

5. Constitutional factors. These may explain a difference between persons in sensitivity to the same stimulus.

6. The character of the sound stimulus—whether continuous or interrupted. If the stimulus is rapidly interrupted but slower than 5 per second, the reflex contractions of the middle ear muscles probably will not be tetanic.

There is evidence that after about forty-five seconds of exposure to a continuous sound the middle ear muscles in man relax with the development of adaptation in the central nervous system. Sound stimuli repeated at slower intervals than this (1 per minute or less) or brief sounds repeated more often will then induce a complete cycle of contraction of the middle ear muscles. Each muscular contraction is preceded by a latent period of about ten and one-half milliseconds,[5] during which time the cochlea is unprotected. This lack of protection of the cochlea is present in some cases of palsy involving the facial nerve to the stapedius muscle. The fatiguing of an ear thus unprotected causes a greater threshold change than does the fatiguing of a normal ear, and a longer period is required for recovery.[6] Furthermore, Finch and Culler[7] have demonstrated that loud sounds interrupted fifty-two times a minute have a much more injurious effect on the hearing of dogs than do continuous sounds of the same intensity. Sounds of this type are produced by rapidly repeating firearms, metal stamping machines and some pneumatic tools. Another important characteristic of sound stimuli is the sharpness of onset. Sounds initiated by explosions, gunfire or the abrupt contact of two metals are due to single exceedingly brief compression pulses of enormous magnitude. The auditory sensation is described as a crack, bang, pop, click, etc. Almost all of the energy in such a pulse corresponds to supersonic or high audible frequencies. A single exposure of the ear to such a stimulus (an explosion, e. g., of gasoline or of a firecracker, as in cases 1 and 2) may cause a permanent loss of hearing for high frequencies. The intensity of explosive sounds is not easily determined. The commonly used noise level meter is unsuited for the measurement of such sounds. Explosions, discharging firearms, pneumatically driven metal tools, metal stamping machines and drop hammers may produce sound stimuli of this nature.

7. The use of protective devices, such as cotton ear plugs and helmets. Dickson and his associates[3] found by testing with the audiometer that aviators wearing helmets escaped injury to the ears.

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5. Perlman, H. B., and Case, T. J.: Latent Period of the Crossed Stapedius Reflex in Man, Ann. Otol., Rhin. & Laryng. **48**:663-676 (Sept.) 1939.

6. Perlman, H. B.: Hyperacusis, Ann. Otol., Rhin. & Laryng. **47**:947-953 (Dec.) 1938.

7. Finch, G., and Culler, E.: Effects of Protracted Exposure to a Loud Tone, Science **80**:41-42 (July 13) 1934.

The depression in the threshold due to occlusion of the canal with cotton wool saturated with petrolatum is as follows:

250 cycles 15 decibels
500 cycles 15 decibels
800 cycles 15 decibels
2000 cycles 15 decibels
3000 cycles 30 decibels
4000 cycles 30 decibels

8. Exposure in an enclosed space or in the open. The use of firearms in a gallery without padding on the walls has been found more injurious than their use in the open. A smooth concrete wall will reflect 96 per cent of the acoustic energy striking it.

9. Previous aural disease. Conduction-deafened ears are probably more resistant to traumatizing sounds than nerve-deafened ears, particularly to air-conducted sounds. Nerve-deafened persons and those with normal hearing will sense the same degree of loudness even for moderately intense sounds. This is not true of persons with conduction deafness.

10. The frequency of the sound. The relation of the frequency of a stimulus to the resultant trauma is not understood. An investigation of this relationship forms the basis of part of this report.

CLINICAL STUDIES

A group of audiograms of persons with acoustic trauma, with a statement of the nature of the acoustic stimulus in each case, follows. The cases may be grouped according to the nature of the acoustic stimulus: (*A*) a single explosive sound, (*B*) rapidly repeated or continuous sound and (*C*) more slowly repeated discrete sound impulses, probably allowing discrete contractions of the middle ear muscles as well as producing a right angle–peaked wave front, with its resultant great transient high frequency stimulus. Many cases encountered which fell in groups *B* and *C* have not been included in this report. The distribution of the cases reported here is as follows:

Group A. Cases 1 to 4.
Group B. Cases 5 to 19.
Group C. Cases 20 to 30.

The audiograms in these cases are shown in figures 1, 2, 3 and 4.

Group A. Single Explosive Sounds.

CASE 1.—Mr. J., aged 28, stated that eleven years prior to examination, when he was 17 years of age, a large firecracker went off near his right ear. He fainted and was dizzy the rest of the day. Since that time he has noted deafness which has improved slightly.

CASE 2.—Mr. W. M., aged 54, had no aural complaints and heard well until Aug. 3, 1937, when a gas tank exploded near him. His left side was exposed to the blast, which knocked him several feet from the spot where he stood. Immediately he noted deafness and tinnitus, which have persisted. Four months later I examined him. Caloric tests produced a normal reaction.

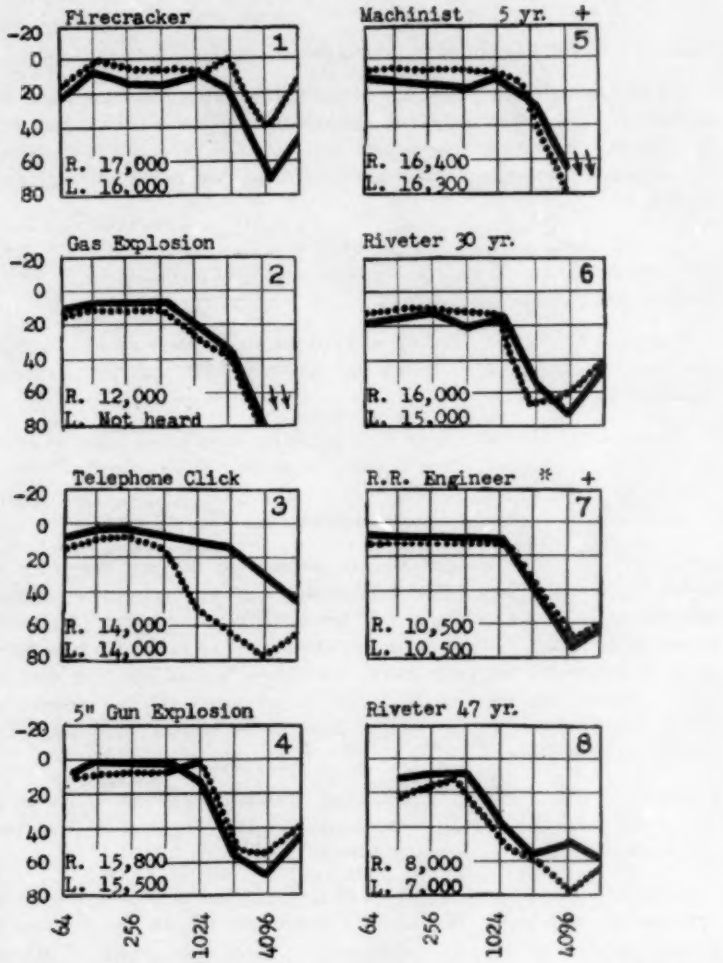

Fig. 1.—The continuous line indicates the hearing in the right ear and the dotted line that in the left ear. The arrows indicate frequencies not heard at maximum intensity of a 2-A audiometer. The figures in the left lower corner represent the upper tone limit for air conduction. R. indicates the right ear and L. the left ear. * indicates an indefinite number of years.

CASE 3.—Mr. A. M., aged 41, worked as a "trouble shooter" for a telephone company and his left ear was exposed repeatedly to sudden loud clicks from a telephone ear piece. For the past year he had noted some hearing impairment but no vertigo or tinnitus.

CASE 4.—In 1919 Mr. S. was stationed on a battleship. He usually wore cotton in his ears when the guns were fired. On one occasion, which he distinctly recalled, a 5 inch (12.5 cm.) gun went off unexpectedly about 10 feet (305 cm.) from him. His ears rang for six weeks after that. Moreover, for eighteen months he was a "second powder man" for a 12 inch (30.5 cm.) gun in the navy. The gun was fired at least once a month, but the noise was less sharp than that from a 5 inch (12.5 cm.) gun.

Group B. Rapidly Repeated or Continuous Sounds.

CASE 5.—Mr. G. H., aged 33, whose father was a blacksmith and had hearing impairment, had been around the blacksmith shop all his life. For several years he worked as a machinist in a very noisy factory. For the past eighteen months he had noted tinnitus and for several years he had noted hearing impairment in the left ear.

CASE 6.—Mr. C. G., aged 44, worked in a steel plant. Since the age of 14 (for thirty years) he had worked in shipyards and steel plants, using an air hammer for riveting and chipping steel.

CASE 7.—Mr. C. T., aged 52, worked for many years as a locomotive engineer and was exposed to loud noises and whistles. He was not aware of hearing impairment.

CASE 8.—Mr. M. Mc., aged 77, worked for forty-seven years as a boilermaker. He first noted deafness about twenty years prior to examination. There was no tinnitus or vertigo.

This case is presented to illustrate extreme acoustic trauma.

CASE 9.—Mr. S. B., aged 31, had worked for the past six years as a motor tester for the air lines. Throughout the working period two airplane motors, mounted on blocks, were run at full speed in the testing room. He could not hear a loud shout in this room, and he wore cotton in his ears. Without the cotton, his ears felt less stuffy but rang more. He devised a cork ear plug with a tiny hole, and the use of this relieved the feeling of pressure. He had a sense of stuffiness and tinnitus in the ears for several hours after leaving the plant. This usually cleared up after sleep.

CASE 10.—Mr. H. L., aged 49, had worked for twenty years at hammering copper and chipping steel with pneumatically driven tools. He had noted tinnitus intermittently, but he experienced little or no hearing loss.

CASE 11.—F. A., aged 55, worked twenty years ago as a riveter in the construction of steel cars. He used no protection for his ears. Twenty to thirty riveters worked at one time about him. There was occasional tinnitus but no appreciable subjective hearing impairment. However, tests showed a definite reduction in hearing of the whispered voice. Since leaving his job as riveter, the patient has worked as a carpenter.

CASE 12.—J. S., aged 27, worked for ten years on stamping machines, stamping out metal parts, such as rivets and screws. In that entire period he had only one week's vacation. Recently, he had noted hearing impairment in the left ear. There was no tinnitus. Tests showed hearing for the whispered voice was reduced in both ears.

CASE 13.—J. P., aged 30, an aviator, worked for a commercial air line for several years. For a six month period he was a motor tester. This work, as that described in case 9, consists of listening to and observing two airplane motors running at full speed throughout each working day. He did not note hearing impairment or tinnitus.

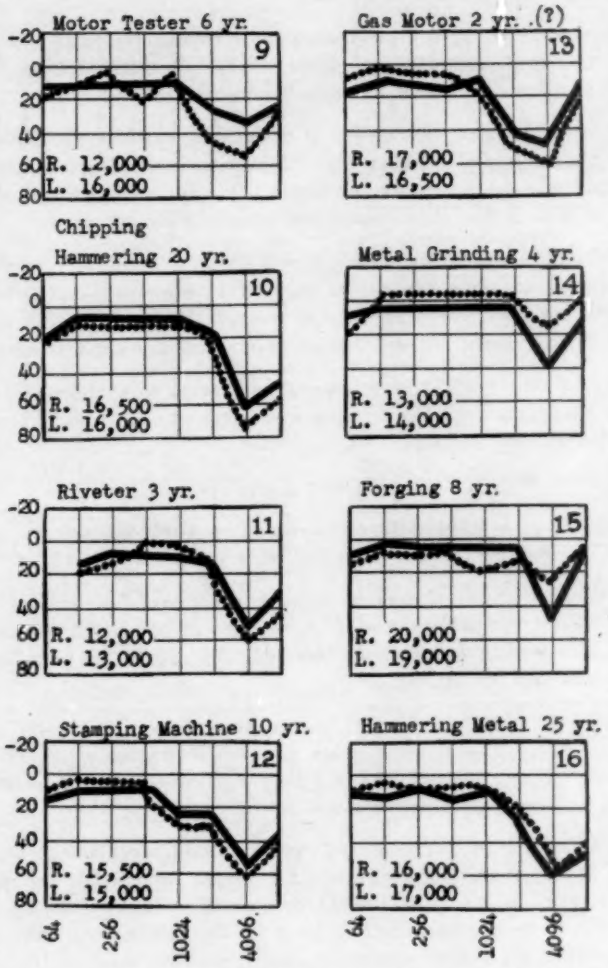

Fig. 2.—The continuous line indicates the hearing in the right ear and the dotted line that in the left ear. The arrows indicate frequencies not heard at maximum intensity of a 2-A audiometer. The figures in the left lower corner represent the upper tone limit for air conduction. R. indicates the right ear and L. the left ear. * indicates an indefinite number of years.

CASE 14.—H. P., aged 26, had worked for four years as a machinist, grinding small tools. There were a number of grinding machines in the same room. His machine was next to one wall of the room, and he attended it in such a manner

that his right ear was exposed to the noise of the other machines in the room, while his left ear was toward the wall. There were no complaints referable to the ear. The audiogram showed a localized dip in the threshold curve for the right ear.

CASE 15.—R. L., aged 26, had worked for eight years in a forging plant, where the noise was so great that only a loud shout near the ear could be heard. There were no complaints referable to the ears.

CASE 16.—J. McC., aged 44, had worked for twenty-five years in a plant where there was continual hammering of metal sheets. Deafness without tinnitus was first noted about two and one-half years before examination.

CASE 17.—L. J., aged 47, worked in a stamping machine factory for eighteen months beginning in 1922. After this the hearing in his left ear was decreased. This decrease was noted only for the watch tick. He stated that all of his friends who have worked in this plant have noted some hearing impairment.

CASE 18.—L. V., aged 52, president of a punch press factory employing forty people, occupied an office which was exposed to the noise, and he was usually seated so that the left ear was directed toward the punch presses. There was occasional tinnitus in the left ear, but no hearing impairment was noted.

CASE 19.—J. Z., aged 38, a painter, a year prior to examination worked for three days inside a boiler while riveting was going on. He said he could not hear for days after leaving the job.

Group C. More Slowly Repeated Sharp Sound Impulses.

CASE 20.—E. S., a 46 year old police officer, used a pistol in target practice and also was a machine gunner in Siberia for three years (1918 to 1921).

CASE 21.—F. H., aged 44, was an engineer in the navy for thirty months during the World War. From the age of 15 to the age of 30 he had done a great deal of hunting with a shotgun and a high-powered rifle. For many years he noted that be heard better with his left ear.

CASE 22.—H. H., aged 48, had been a trap shooter for many years, until twenty years ago. Twenty-eight years ago, for three two week periods a year apart he had artillery practice with a 3 inch (7.5 cm.) gun. He wore no cotton in his ears. No hearing impairment was noted.

CASE 23.—Mr. C. M., 47 years old, was in the Italian artillery for two years during the World War. Tinnitus had been present for the last few years. This resembled a 4096 cycle tone and could be induced by tightly closing the meatus with a finger or by pressing the ear piece of the audiometer firmly against the ear. No hearing impairment was noted.

CASE 24—D. S., aged 48, a policeman, was in active service during the World War, and since then he had done target practice regularly in a bare-walled basement room. He fired about one hundred and twenty rounds every three months. No hearing impairment or tinnitus was noted.

CASE 25.—A. N., aged 62, had worked as a machinist all his life. For the past fourteen years he had worked in a mower plant, using a trip hammer and machines of other types. The noise was so great that only very loud speech was heard. There were no aural complaints.

CASE 26.—T. B., aged 38, had done a great deal of hunting and skeet shooting. Tinnitus had been noted for the past three years and was aggravated by the firing of a gun and other loud sounds. There was greater depression in hearing on the left. This corresponds to the observations of others with respect to the firing of guns from the right shoulder and the exposure of the left ear to the explosive

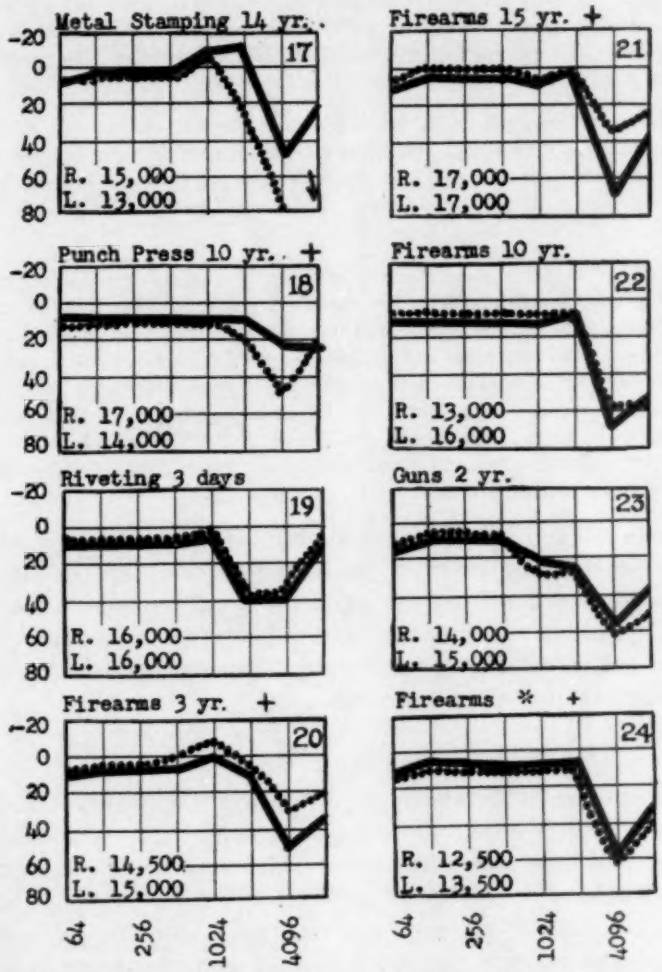

Fig. 3.—The continuous line indicates the hearing in the right ear and the dotted line that in the left ear. The arrows indicate frequencies not heard at maximum intensity of a 2-A audiometer. The figures in the left lower corner represent the upper tone limit for air conduction. R. indicates the right ear and L. the left ear. * indicates an indefinite number of years.

sound at the end of the gun barrel. The head acts as a shield for high frequencies and thus protects the opposite ear. At 6000 cycles the shielding effect is 25 decibels.

CASE 27.—G. S., aged 20, worked in a dynamite crew for a month in 1937 but wore cotton in his ears. In 1936 he worked for six months in a forging plant,

where there was a great deal of hammering on anvils. He did not protect his ears. There were no aural complaints.

CASE 28.—D. S., aged 47, for about twenty years had noted impaired hearing when attempting to listen to a group of people. The deafness was not progressive and there was no tinnitus. The patient dated his hearing difficulty to service in the World War. For sixty-six consecutive days he served in a crew firing a French "75." While he usually protected his ears with cotton, on at least six occasions he failed to use cotton. He was particularly deafened for a day or two after each of these occasions.

CASE 29.—B. S., aged 38, in his work used his left ear for the telephone at least one hundred times a day. He often received loud shocks through the receiver. For the past ten years he had been doing target practice in a bare-walled basement room, with a 45 caliber revolver. There were at least six periods of target practice a year. There were no aural complaints.

CASE 30.—This case illustrates the value of a continuous frequency oscillator in studying and detecting dips in the threshold curve. F. M., aged 28, had been in the National Guard for four years. For ten days each year he fired a 75 mm. cannon fifteen to twenty times a day. He also fired it once a week throughout the year. He was stationed about 10 feet (305 cm.) behind and to the left of the gun. An audiogram made with a continuous frequency oscillator shows a narrow dip in the threshold curve for his right ear—between 4096 and 9474 cycles, maximum at about 7000 cycles. This dip in the audiogram would have been missed if only octave frequencies had been used.

Dips in the audiogram in acoustic trauma need not be maximum at 4096. The shielding effect of the head for these high frequencies is sufficient to protect the left ear when the subject stands to the left of the gun. Further investigation of threshold dips in acoustic trauma, with the aid of the continuous frequency audiometer, should increase knowledge of this otologic problem.

EXPERIMENTAL STUDIES

A group of cases such as those just presented, gathered from a large number in which mixed otolaryngologic signs and symptoms were observed, add little more than mass evidence of the clinical entity of acoustic trauma. A number of more controlled observations that help to clarify this condition have been reported. One type of controlled study is the audiometric examination of a group of workers in a single industry, as reported by Larsen [2] and by a number of industrial commissions referred to in the paper of McCord and his associates.[4] Another type is the controlled animal experiment. In this type, auditory acuity may be tested, as reported by Finch and Culler,[7] or histologic changes in the cochlea may be noted. A third type is the study of the response of subjects with normal hearing to the acoustic trauma of a specific industrial noise, as reported by Larsen [2] for riveting and by

Dickson and his associates [8] and by Campbell and Hargreaves [8] for airplane noise. The changes in auditory threshold revealed in the last-mentioned type of study were temporary but need to be considered only as a difference in the degree of injury. A fourth type of observation, carried out in the field of psychology and reported by Ewing and Littler,[9] Rawdon-Smith,[10] Parker [11] and others, concerns the effect of loud pure tones of short duration (i. e., two minutes) on the auditory threshold of normal young adults. Numerous observa-

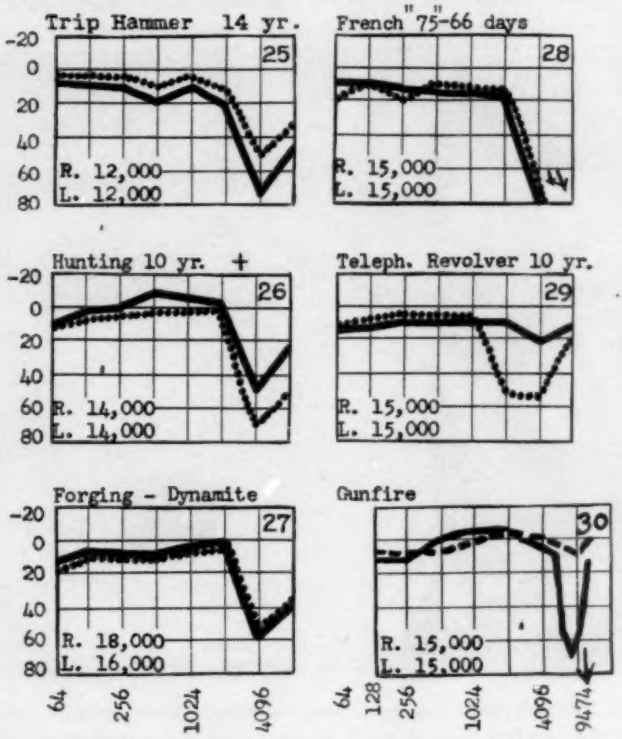

Fig. 4.—The continuous line indicates the hearing in the right ear and the dotted line that in the left ear. The arrows indicate frequencies not heard at maximum intensity of a 2-A audiometer. The figures in the left lower corner represent the upper tone limit for air conduction. R. indicates the right ear and L. the left ear. * indicates an indefinite number of years.

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8. Campbell, P. A., and Hargreaves, J.: Aviation Deafness—Acute and Chronic, Arch. Otolaryng. **32**:417-428 (Sept.) 1940.

9. Ewing, A. W. A., and Littler, T. S.: Auditory Fatigue and Adaptation, Brit. J. Psychol. **25**:284, 1935.

10. Rawdon-Smith, A. F.: (*a*) Auditory Fatigue, Brit. J. Psychol. **25**:77-85 (July) 1934; (*b*) Experimental Deafness, ibid. **26**:233-243 (Jan.) 1936.

11. Parker, R. C.: The Nature of Fatigue on the Auditory System, Proc. Physiol. Soc. **50**:108-118, 1938.

tions of this kind were made in the laboratories of the division of otolaryngology at the University of Chicago as a preliminary to the study reported in this paper. These observations, with observations on the middle ear muscle reflex, form the basis of a report to be published on central and peripheral fatigue in the auditory apparatus. Furthermore, they have given information on the nature of tinnitus produced by loud tones. Unfortunately, the threshold changes resulting from short stimuli are so fleeting (i. e., forty-five seconds) that a complete threshold curve cannot be obtained for any single exposure. In spite

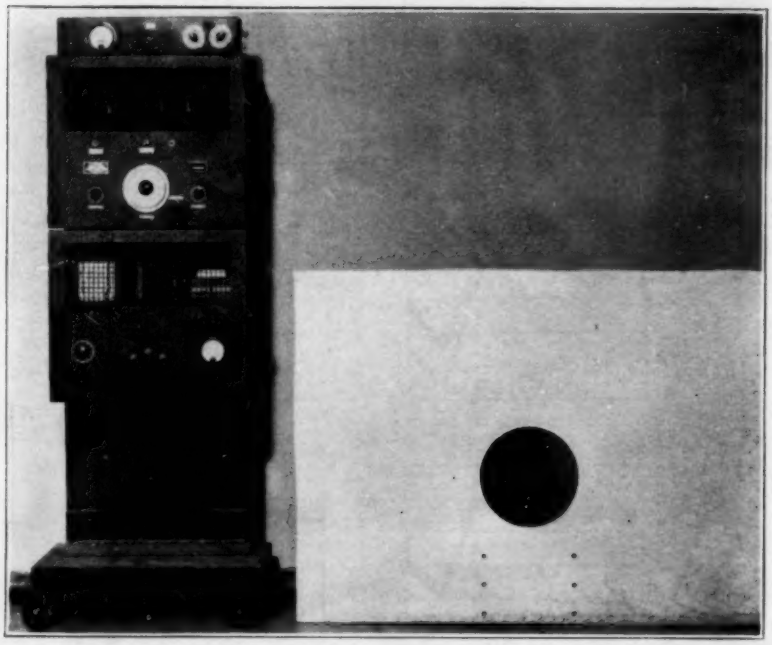

Fig. 5.—The apparatus used to produce very loud pure tones in a study of the relation of the frequency of acoustic trauma to the resultant depression in the threshold curve.

of all these investigations, the reason for a localized depression of the auditory threshold curve at 4096 cycles as an early sign of acoustic trauma remains unexplained. Larsen [2] considered the following possibilities in discussing this point:

1. There may be a greater sensitivity of normal ears to frequencies between 3000 and 4000 cycles.

2. The observation may be related to the resonant frequency of the external auditory canal, which is about 3000 cycles.

3. Tinnitus may interfere with threshold determinations.

4. The 4096 dip may be unrelated to the frequency of the fatiguing sound, since analysis of the sound spectrum of some of the noises known to cause injury (riveting) revealed that the frequencies of maximum intensity were at least an octave below 4096 cycles.

5. Since the circumscribed defect has been found in conditions other than professional deafness, such as intoxication, syphilis, trauma to the head and retinitis pigmentosa, Larsen,[2] after disposing of the other factors mentioned, wrote: "the place of perception for C^5 (4096) presents a special vulnerability, decreasing towards both sides." He said he thought this might be due to poor blood supply, resulting from the bifurcation of the cochlear ramus of the auditory artery at this point in the cochlea (basal coil) or to a narrowing of the cochlear duct here.

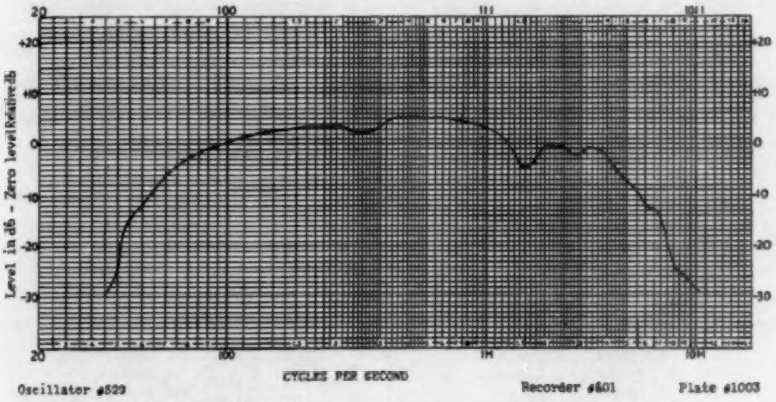

Fig. 6.—Frequency response curve of the loud speaker used in producing acoustic trauma. Note the uniform response from about 70 to 5000 cycles.

Further attempts to explain the 4096 dip observed in the early stages of acoustic trauma and a general investigation of the relation of the frequency of the fatiguing tone to the shape of the resultant threshold curve and to the upper tone limit seemed indicated. The effect of very loud pure tones on the threshold curve therefore was reinvestigated.

Procedure.—An apparatus was used which is capable of producing intense and relatively pure sounds (analysis in table 1) of any frequency tested by the 2-A audiometer.[12] After an hour's exposure to such a sound the depression in the threshold curve was usually sufficiently lasting to permit repeated audiograms. Of the eight frequencies produced by the 2-A audiometer, all but one (128 cycles) were used. With a uniform time of exposure (one hour) and approximately uniform loudness (near the threshold of pain—table 2) of the fatiguing tone, information was obtained as to the fatiguing effect of each frequency and the type of threshold curve each frequency produces. Three to 6 normal young adult subjects were used for each frequency studied. One ear was plugged with petrolatum-

12. A continuous frequency audiometer was not available for most of the threshold studies; hence, the hearing for frequencies between octaves was not determined.

treated cotton, not only as a precautionary measure but in order that the effect of this type of aural protection might be noted. The apparatus is illustrated in figure 5. The baffle board on the loud speaker improved the output and quality of low frequency sounds. The apparatus was used in a large bare-walled room in the basement. This room contained a number of panels on which were mounted large electric switches. In a general way the conditions existing in many factory operations were simulated. All the subjects were placed in the same position with reference to the loud speaker, but this probably was not important, particularly for frequencies under 2048, since the shielding effect of the head is not great (10 to 15 decibels) for those frequencies and since the reflection from the walls was so great for all frequencies used. Audiograms and monochord tests were made imme-

TABLE 1.—*Output of the Oscillator at the Maximum Intensity for Four Frequency Settings Studied with a Wave Analyzer Measured in Millivolts and Representing One Tenth of the Output of the Oscillator*

64 Cycles		Output, 1830	256 Cycles		Output, 1860	1024 Cycles		Output, 2000	2048 Cycles		Output, 1850
128		52	512		52	2048		128	4096		185
192		37.5	768	Har-	83	3072		165	6144	Har-	172
220	Har-	13.8	1280	monics	136	4096	Har-	84	8192	monics	150
256	monics	2.5	4096		2.5	5120	monics	86			
360		140				7168					
4096		0									

TABLE 2.—*Intensity of Fatiguing Tones Three Feet from the Loud Speaker Measured in Decibels from 10^{-16} Watts per Square Centimeter with a General Radio Noise Level Meter* *

64 cycles	102 decibels
256 cycles	98 decibels
512 cycles	118 decibels
1024 cycles	90 decibels
2048 cycles	106 decibels
4096 cycles	108 decibels

* These sound levels are comparable to those prevailing in a boilerworks.

diately before and immediately after exposure to the sound stimuli. Subsequent audiograms were made until there was complete recovery.

In a study of this type the purity of the sound stimulus is important and is attested to by the frequency response of the loud speaker (fig. 6) and by the analysis of the output of the oscillator with a wave analyzer (table 1). It is seen that the output of the loud speaker is quite uniform up to about 6000 cycles. At 8000 cycles there is a sharp drop. The largest harmonic output of the oscillator at maximum intensity is less than 10 per cent of the fundamental. The intensity of the sound was measured in decibels from a commonly used reference level (10^{-16} watts per square centimeter) with a General Radio noise level meter and was close to the threshold of pain. The intensity for each frequency is recorded in table 2. In reviewing the results, each frequency may be considered separately. The audiograms resulting from exposure to different frequencies appear in figure 7.

64 Cycles.—Three subjects were exposed. Their left ears were closed with petrolatum-treated cotton. The intensity of sound was great enough to mask a loud shout at the ear and even made it impossible to hear one's own voice. Toward the end of an hour's exposure a tickling sensation was experienced in the right, or unprotected, ear. The sound in the right ear appeared impure, because some high overtones were sensed deep in the ear.

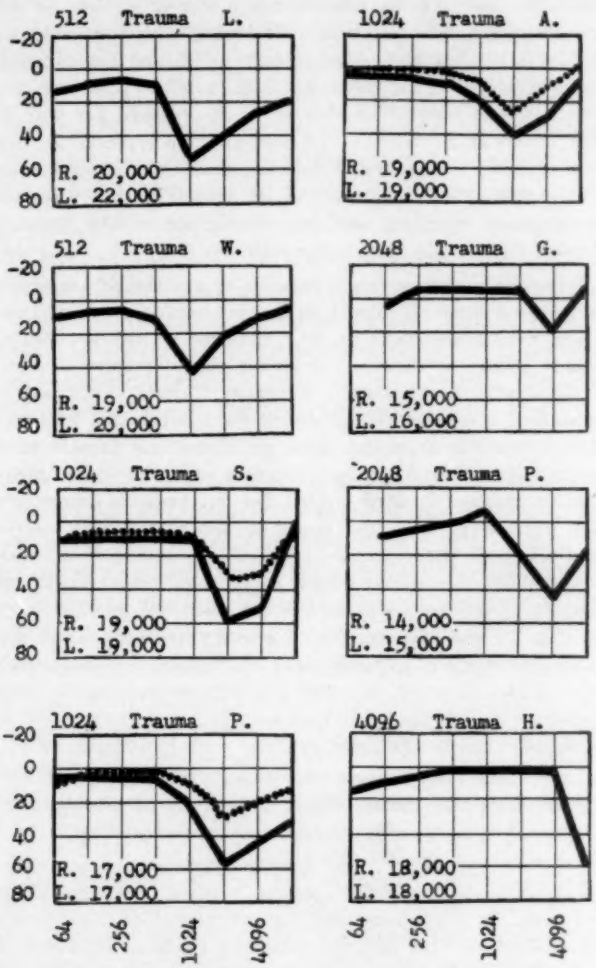

Fig. 7.—Audiograms made after one hour's exposure to single frequencies near the threshold of pain (about 100 decibels from 10 to 16 watts per square centimeter).

Audiograms were made immediately after the subjects left the testing room or within five minutes. No alteration from the normal threshold curve was observed. There was no change in the upper tone limits.

256 Cycles.—Six subjects were exposed in two different periods. The loudness of the sound was sufficient to mask a loud voice at the ear. The left ear was protected with petrolatum-treated cotton and showed no acoustic trauma. No con-

sistent results were obtained for the right ear. One subject showed no effect, 3 showed a dip at 256 cycles, 1 showed a dip at 4096 cycles and another a dip at 512 cycles. Tinnitus was experienced for a short period by 2 subjects at frequencies varying from 512 to 2048.

512 Cycles.—Three subjects were exposed to a tone sufficiently loud to mask a loud spoken voice at the ear. After an hour's exposure there was little or no effect in 1 subject. None of the subjects had a loss in the left, or protected, ear. Two subjects showed a definite dip at 1024 cycles, with recovery in about forty-eight hours. In 1, tinnitus was observed only on closing the exposed ear with a finger and was matched in the other ear with a 1024 cycle tone at 25 decibels. In the other 2 subjects tinnitus on closure of the exposed ear was matched with a tone of 2048 cycles at 30 decibels. In both of these subjects the tinnitus was of short duration and had disappeared before a normal audiogram was obtained. It did not interfere with the final results of the threshold tests. Immediately after exposure, diplacusis to whistling was experienced, the whistle sounding an octave higher in the left (protected) than in the right ear.

1024 Cycles.—Three subjects were exposed to a sound of an intensity near the threshold of pain. A sense of fulness in the ear, headache and dull pain about the unprotected ear were experienced by all. Cupping of the ear with a hand, with resultant sudden change in intensity, caused a lateral pulsion of the head in the direction of the source of the sound. There were slight ataxia and nausea when the subjects walked about the room. In all the subjects the left (protected) ear was less affected than the right. A sense of fulness was present in the right ear for over twelve hours after exposure. Tinnitus appeared to be about 1024 cycles and in 1 subject changed to 2048 cycles, but in threshold observations the frequencies from 1024 to 8192 cycles seemed to appear suddenly on the sensorium and were distinct from the tinnitus. One subject described a hissing sound in addition to tinnitus. Tinnitus and fulness disappeared before the threshold became normal. Closure of the ear or coughing increased the tinnitus or caused it to recur again after it had disappeared. Normal thresholds were obtained about seventy-two hours after the exposure was terminated. A disagreeable sense of confusion due to the distortion of commonly experienced sounds, such as speech, telephone conversation and the subject's own voice, was present for several hours after the exposure. There was some difficulty with localization of sound.

2048 Cycles.—Three subjects were exposed to a stimulus of loudness comparable to that of lower frequencies. One subject had a drop of 15 decibels at 4096-8192. The other 2 showed a dip at 4096. None of them had tinnitus. The left ear was not affected. Recovery occurred in forty-eight hours.

4096 Cycles.—The exposure of 3 subjects resulted in a sense of fulness in the ear in all of them. In 2 subjects mild tinnitus resembled a pitch of 8192 cycles. In the third, tinnitus of 8192 affected threshold readings on the first test. The left ear was unaffected. In all the subjects the drop in threshold occurred at 8192.

8192 Cycles.—In the exposure of 2 subjects, no effect on the threshold curve or on the upper tone limit was noted.[13]

COMMENT

Military Aspects.—Many of the acoustic stimuli which produce trauma to the auditory apparatus are in the nature of brief sound pulses

13. The loud speaker is considerably less efficient at 8000 cycles than at the frequencies below this level.

(gunfire, explosions, noises produced by punch presses and forges, etc.). It is well, therefore, before examining the results of exposure to a continuous tone, to review what is known of the behavior of the ear in the presense of brief sound pulses.

As has been pointed out in the beginning of this report, frequently repeated sharp brief loud sounds induce a corresponding series of contractions of the middle ear muscle. Before each contraction there is a latent period, during which the vibrations of the ossicular chain are not dampened by the contracting muscles. During this period (10.5 milliseconds) the cochlea is unprotected and hence more violently stimulated. In certain cases of facial paralysis examination of the unprotected cochlea after a brief exposure to a loud tone reveals a greater degree of depression in the threshold curve and a longer period of recovery than on the normal side. In this way one can explain the fact that the traumatizing effect from frequently repeated sharp sounds is greater than that from a continuous sound of similar intensity and duration.

The nature of the physical forces developed by an explosion or in gunfire deserves special consideration, so that one may better understand how these forces may injure the auditory apparatus. Until recent years little was known about their nature. Little is yet known as to how they stimulate the ear and produce the auditory sensations associated with them. During and after the World War, with the development of special recording and measuring instruments and under the direction of military services, considerable information was obtained. In this country the intensity and propagation of shock waves developed by an explosion of TNT and by gunfire has been studied by Thompson [14] at the Naval Proving Ground, Dahlgren, Va. The pressure acoustic phenomena near a discharging rifle have been extensively investigated with the phonodeik by the late Prof. D. C. Miller,[15] of the Case School of Applied Science, Cleveland.

The shock pulse produced by detonation of an explosive may be defined as a sound wave of great condensation with a great initial velocity. The velocity of propagation near the source is much greater than the normal velocity of sound but is rapidly reduced to that of sound. The intensity of the shock wave 26 feet (790 cm.) from an explosion of 10 pounds (4.5 Kg.) of TNT was computed by Thompson to be 10^4 watts per square centimeter, about 80 decibels above the threshold of feeling, or 200 decibels above the threshold for hearing of periodic sound. Because the duration of this pulse is exceedingly short, the ear is less affected than when it is exposed to a sustained

14. Thompson, L.: Shock Waves in Air and Characteristics of Instruments for Their Measurement, J. Acoust. Soc. Am. **12**:198-204 (July) 1940.

15. Miller, D. C.: Sound Waves: Their Shape and Speed, New York, The Macmillan Company, 1937.

sound of much less intensity—but in its propagation and dissipation the shock pulse intimates molecular displacements to which the cochlea reacts more violently and before the middle ear muscles can contract. Miller's observations are particularly useful in understanding what the ear is exposed to in gunfire. The record of the sound from a 12 inch (30.5 cm.) rifle, taken from a position 1,500 feet (457 meters) in the rear of the gun, shows a positive pulse (compression) rising abruptly to a maximum and falling to a negative pressure (rarefaction). The intensity of the negative pressure is usually much less than that of the compression pulse, but it continues for a long time. Windowpanes may be cracked by the positive pulse, and the fragments (before they can fall inward) are pulled outward by the more prolonged negative pulse that immediately follows. The positive pressure pulse may be strong enough to rupture the drum membrane.

The maximum pressure recorded with the phonodeik at this distance is 2.6 Gm. per square centimeter and occurs about fifteen thousandths of a second after the beginning of the sound pulse. The pressure becomes normal in twenty-five thousandths of a second and then becomes negative for five hundredths of a second or longer. Translated into acoustic pressure units it is very large (2.6 Gm. is equivalent to about 2,368 dynes per square centimeter). Because of the inertia of the recording device, the slope of the pressure pulses recorded with the phonodeik is probably much sharper than the record indicates.

Representative of other observations by Miller on the acoustic pressure pulses of gunfire are those made from a station located 3,000 feet (915 meters) in front of a 14 inch (35.5 cm.) gun. Sound effects called the "crack" and the "boom" of the gun were recorded. The crack comes only from the projectile and is due to two sharp compression pulses about two hundredths of a second apart, with a negative pulse between them and followed by irregular shallow waves. The main pulses are due to the sound waves developed at the point and base of the projectile, while the irregular waves are produced by the turbulence left by the projectile as it plows through the air.

The boom coming from the exploding gas at the muzzle of the gun consists of a short positive pulse, amounting at this distance to a compression of about 2.2 Gm. per square centimeter, which lasts for fifteen thousandths of a second and is followed by a rarefaction of less than 0.5. Gm. per square centimeter, which lasts about one tenth of a second. The crack is heard first, because it travels with an initial velocity equal to that of the moving projectile (2,600 feet [793 meters] per second). The boom, traveling with a mean velocity of sound (about 1,120 feet [336 meters] per second), arrives later, and when the observer is 3,000 feet (915 meters) in front of the gun it is heard about one second after the crack (figs. 8 and 9).

Miller determined the pressures prevailing at various points near a discharging gun. Maximum pressures (incompatible with life) are recorded in front of the muzzle, varying with the elevation of the gun and its size (i. e., 38,200 Gm. per square centimeter or 542 pounds

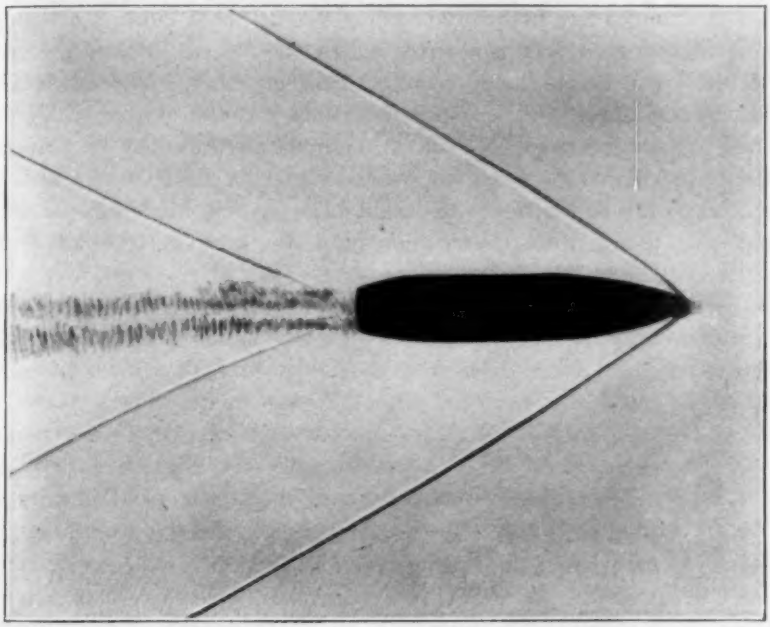

Fig. 8.—Sound waves produced by a bullet moving with a velocity of 2,700 feet (823 meters) per second. These waves from the nose and base of the bullet cause the "crack" heard by an observer stationed at some distance from the gun and are recorded in the first part of the tracing in figure 9. From Miller,[15] by permission of The Macmillan Company, publishers.

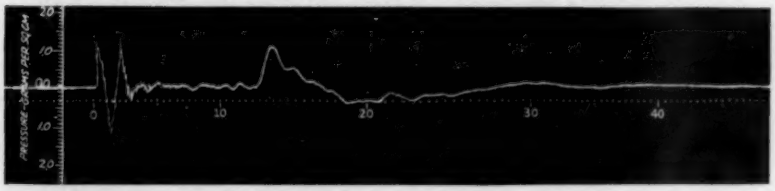

Fig. 9.—Phonodeik record of the shell and gun waves from a 12 inch (30.5 cm.) rifle. Two positive pulses immediately after zero time with a negative pulse between them come from the nose and base of the shell and are heard as a "crack." The slower pulse after 10 seconds comes from the exploding powder in the gun barrel and is heard as a "boom." This record was taken from a position 600 feet (183 meters) in front of and about 300 feet (91.5 meters) to the side of the gun. From Miller,[15] by permission of The Macmillan Company, publishers.

per square inch on the ground 12 feet [3.5 meters] in front of the muzzle of a 12 inch [30.5 cm.] gun). While the pressures about the breech of the gun, where the men are usually stationed, are much smaller, for example, 100 Gm. per square centimeter, they are enormous when translated into acoustic pressure equivalents.

The firing of any firearm will set in motion these pulses. A subject who is near a gun when it is fired will not be able to identify the crack and the boom, because they occur so close together. Nevertheless the auditory apparatus will be exposed to their trauma. Other, more sustained acoustic phenomena, such as might be produced by resonance of the gun barrel and mountings or vibrations of the gas column within the gun barrel, are so much smaller in intensity as to contribute a relatively small part to the acoustic sensation or to the acoustic trauma. These single, exceedingly short pressure pulses are difficult to record accurately, because of distortion and resonances in all recording systems. Using an improved recording system, Thompson [15a] obtained an almost right angle wave front for a pulse from a powder blast of a medium caliber gun. The pulse was recorded with the gage in the muzzle plane and 14 feet (4 meters) from the axis of the barrel. The lapsed time from the onset of the pulse to the return to the base line was about five tenths of a millisecond. The cochlea reacts to pressure disturbances of that duration.

The smaller and more slowly rising pulses which develop at some distance from a large gun are less loud and cause less acoustic trauma than the more rapidly rising and enormous pressure pulses which develop near these guns. Proximity to small firearms exposes the ear to smaller but similar pressure pulses, the acoustic pressure pulses being of great intensity as compared with the limit of hearing of the normal ear. Any wave front, even these nonperiodic pulses, can be analyzed into its frequency components. A slowly rising front contains a greater proportion of its energy in low frequencies. A sharply rising wave front has a greater proportion of its energy corresponding to high frequencies. The pulse waves are so steep as to have much of their energy corresponding to the supersonic range of frequencies. Their effect on the cochlea is unknown. The cochlea acts essentially as a harmonic acoustic analyzer and responds to the pressure pulses accordingly. A steeply rising wave front will probably cause a greater irritation of the cochlea near the round window, where high frequencies are perceived. A slowly rising acoustic pulse will probably cause maximum irritation of the portion of the cochlea near the apex, where low frequencies are perceived. Of course, if the pulse wave rises more slowly than a sinusoidal acoustic wave at the lower tone limit, it will not produce a localized stimulus in the cochlea, no sound will be perceived and no trauma to the cochlea will occur. Since low frequency sounds are less traumatizing than higher

15a. Personal communication to the author.

frequency sounds, the sharper the pulse rise, everything else being equal, the greater the acoustic trauma.

When small arms are fired in a bare-walled room, the enormous acoustic pressure pulses originating from the nose and base of the bullet and from the explosion of gas at the muzzle are reflected many times to the subject's ears, with little loss of energy. A smooth concrete wall reflects 96 per cent of acoustic energy striking it. The resultant enormous increase in the number of pulses which the ear is exposed to causes more rapid acoustic trauma than when these firearms are used in the open. Both ears will be equally traumatized by the reflected waves. The shielding effect of the head for high frequencies is not a factor, since the acoustic pulses are reflected from walls on both sides of the subject.

A great variety of observations on the response of the ear to a continuous sound must be reviewed when one attempts to evaluate the results of the acoustic trauma experiment reported in this paper. They may be grouped under different headings.

Masking.—While the ear is hearing one sound, it is less able to hear others. The degree of masking can be defined as the depression of the threshold in decibels for one tone due to the presence of the masking tone. Low frequencies have a greater masking effect than high frequencies. For great intensities this masking effect spreads over a wide area of the auditory spectrum—peaking at the frequency of the sound used and its harmonics. The phenomenon of masking has been localized in the cochlea by Derbyshire and Davis,[16] who have shown that action potentials from the auditory nerve in response to a click can be reduced in size by the addition of a pure tone. Kemp, Coppee and Robinson[17] observed similar responses from the lateral lemniscus. From the masking characteristics of audition, if considered from the standpoint of disturbances in the cochlea, one would expect a more widespread drop in the threshold curve on exposure to single frequencies than was observed in this investigation. This discrepancy between the widespread masking effect of a loud pure tone and the localized threshold changes resulting from exposure to this tone may find its explanation in the peculiarities of innervation of the outer hair cells. While the inner hair cells each receive a single fiber, the fibers going to the outer hair cells course downward toward the round window, establishing connections with large numbers of outer hair cells (over half a turn of the cochlea). A small locus of stimulation from a single masking frequency may interfere with the response of large numbers

16. Derbyshire, A. J., and Davis, H.: The Action Potentials of the Auditory Nerve, Am. J. Physiol. **113**:476-504 (Oct.) 1935.

17. Kemp, E. H.; Coppee, G., and Robinson, E. H.: Electric Responses of the Brain Stem to Unilateral Auditory Stimulation, Am. J. Physiol. **120**:304-315 (Oct.) 1937.

of outer hair cells below this level. This may explain the widespread masking effect of a loud pure tone and the fact that a low tone masks a wider band of the acoustic spectrum than does a high tone. As the outer hair cells above the locus of disturbance by the masking tone are not affected by the collateral innervation, and since they would respond to the lower tones, frequencies below that of the masking tone are much more difficult to mask than frequencies above that of the masking tone. Therefore, while the masking effect is widespread, the maximum stimulation and trauma to hair cells are probably much more localized, resulting in a much more localized depression of function. On the other hand, observations on masking, with the middle ear muscle reflex used as an index of activity of the lower auditory system (cochlea, primary cochlear nucleus, superior olive), indicate that the phenomenon of masking is probably located in the higher centers. These observations, made in the laboratory of the division of otolaryngology at the University of Chicago, appear as a part of a separate report now in press.

Fatigue.—Studies of auditory fatigue in man previous to those reported here have been carried out with relatively weak stimuli, causing brief and slight changes in function. On the other hand, experiments of this type on animals have entailed the use of intense prolonged stimuli and have resulted in a widespread permanent depression of function and in evidence of local disruption of the organ of Corti. The localization of the reversible phase of acoustic trauma is difficult. Observations on the action potentials of the auditory nerve may be mentioned here. The onset of a tone stimulus induces a sharp spike of action potentials which fall rapidly in intensity (the greatest change occurs within two seconds) but continue at the lower level, presumably for the duration of the stimulus. Thirty seconds after the tone is stopped, recovery is complete. This characteristic response of the auditory nerve to sound stimuli may be considered a special form of fatigue. But it probably represents a new level of sustained activity and not an exhaustion of reserve material. Rawdon-Smith [10b] has indicated that the higher centers are involved in auditory fatigue by demonstrating that momentary darkness had a disinhibitive effect on the auditory threshold depressed by a fatiguing stimulus, which tended to restore the threshold to the normal. Similar observations of subjects under hypnosis were made by Nemtzova.[18] Observations made in this laboratory on fatigue in the lower centers subserving audition, as reflected in the acoustic contractions of the middle ear muscle, also indicate that fatigue from moderately intense acoustic stimuli (comparable to those

18. Nemtzova, O.: The Influence of the Central Nervous System upon Some Physiological Processes During Work: The Changes of the Auditory Threshold, Bull. biol. et de méd. expér. URSS **1**:426-427, 1936.

previously used in experiments on man) cannot be demonstrated in the lower auditory centers. Intense stimuli (comparable to those used in this study) do involve the lower centers as tested by this means. The location and nature of the irreversible lesion following acoustic trauma are suggested by the work of Crowe, Guild and Polvogt.[19] They studied many subjects who had an abrupt high tone loss (above 1000 or 2000 cycles), and found atrophy of the auditory nerve and degeneration of the organ of Corti limited to the basal coil of the cochlea.

Relation of the Frequency of the Fatiguing Tone to the Resultant Threshold Curve.—The ear does not distort pure tones of low intensity, but when it is stimulated by a loud pure tone, one hears not only the tone but a series of harmonics (subjective tones). This is largely due to the nonlinear response of the conducting mechanism. A harmonic can be detected by introducing a variable searching tone that can be made to beat with it. Low tones produce harmonics at a lower level of intensity than high tones. Above 1000 cycles the intensity must be raised above 50 decibels to produce the first distortion (second harmonic). The intensity of the harmonics (subjective tones) is considerable. The intensity of two subjective tones (2400 and 3600 cycles) arising from a 1200 cycle tone at 80 decibels are 60 and 50 decibels, respectively. Bronstein and Churilova[20] and Rawdon-Smith[10] observed that high frequencies have a greater fatiguing effect than low frequencies. Rawdon-Smith found that for high frequencies (1000 and 2000 cycles) temporary deafness often involved tones an octive above the fatiguing tone. Since the ear is more sensitive to the higher frequencies of the overtones (when 1000 cycles and frequencies below this are used for the stimulation tone), these harmonics (subjective tones) may have a greater or as great a traumatic effect on the ear as the stimulating tone. This may account for the fatiguing effect observed by him at these higher frequencies but does not explain why the audiograms I obtained show a relative lack of fatigue (drop in the threshold curve) at the level of the stimulating tone as compared with the octave above it. From available information I am unable to explain the occurrence of the maximum depression in the threshold an octave above the frequency of the fatiguing tone. S. S. Stevens,[20a] of the phychological laboratory of Harvard University, has also observed that the maximum dip in the audiogram is above the frequency of the fatiguing tone. He found that the maximum dip was not ordinarily

19. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness, Bull. Johns Hopkins Hosp. **54**:315-379 (May) 1934.

20. Bronstein, A. J., and Churilova, E. A.: The Dependance of Time Taken to Restore the Initial Excitability of the Hearing Apparatus upon the Pitch of the Acting Tone, Bull. biol. et méd. expér. URSS **1**:428-430, 1936.

20a. Stevens, S. S.: Personal communication to the author.

at the next octave unless the ear is tested only at the octave frequency. Testing with a continuously variable frequency, he found the maximum loss ordinarily to be somewhere near the middle of the octave, just above the fatiguing tone. He could not explain this.

He found further that when the ear is exposed to a continuous flat sound spectrum a dip in the audiogram occurs only in the high frequency region. This dip is usually most marked at frequencies near 5000 cycles per second.

SUMMARY OF OBSERVATIONS

When acoustic trauma was produced (depression in the threshold curve), the maximum dip was usually an octave above the frequency of the fatiguing tone. This was true for 512, 1024, 2048 and 4096 cycles. The results were not uniform for 256 cycles and a test could not be made for 8192 cycles.

Therefore a definite relation is found between the frequency of the stimulus and the resultant audiogram, but the greatest depression was not (as one would expect) at the frequency of the fatiguing tone.

A uniform threshold curve (i. e., with a 4096 cycle dip) was not obtained for a series of tones of approximately uniform intensity used to produce acoustic trauma.

A threshold curve commonly found in early acoustic trauma (with a localized 4096 dip) was obtained in subjects exposed to a fatiguing tone of 2048 cycles.

Sounds of low frequency (64-256) produced less acoustic trauma than those of higher frequency and of approximately equal intensity.

Exposure to a fatiguing tone of 4096 cycles did not produce a threshold curve with a localized depression at 4096 cycles but produced a curve with a localized drop for the tone an octave above it (8192 cycles).

The upper tone limit, as tested with the Struychen monochord, was not affected by any fatiguing tone used.

Petrolatum-treated cotton provides limited protection from acoustic trauma. For the stimulus used, complete protection was obtained for all but 1024 cycles. At this frequency the protection was definite but only partial.

Tinnitus following exposure was of variable duration, and the frequency of the tinnitus was not regularly identified with the frequency of the fatiguing tone but often was an octave above it.

Tinnitus thus produced was uniformly increased in intensity by closure of the external auditory canal. When tinnitus had stopped, it could be made to reappear by closing of the auditory canal with a finger. Tinnitus usually stopped before the threshold curve became normal. The values of the threshold determinations were thus little interfered with.

MULTIPLE MYELOMA WITH LARYNGEAL METASTASIS

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To von Rustizky (1873) goes the credit for proposing the name "multiple myeloma" for the clinical entity originally described by William McIntyre in 1850 [1] as "mollitis ossium." An intimation that the disease existed centuries ago is brought out by Ritchie and Warren,[2] who discovered the skeleton of an American Indian dating from the latter part of the ninth century which showed widespread destructive lesions of the bones very suggestive of this disease.

Multiple myeloma is relatively rare. Prior to 1937, 643 cases were recorded in the literature.[3] Reports of this disease appeared with the same degree of frequency in all parts of the world. At Johns Hopkins Hospital [4] this pathologic condition was observed in 4 of 9,000 autopsies, and life insurance tables give its incidence as 0.03 per cent of all malignant growths.[5]

Multiple myeloma is a disease of later life, a large percentage (80) of the patients being from 51 to 60 years of age. Coley [6] stated the opinion that isolated reports of such growths in young adults and children in all probability fall under the classification of endothelial myeloma or Ewing's sarcoma with generalized involvement. His series of cases contains 1 in which a tumor in a boy aged 17 proved to be multiple myeloma and Slavens [7] reported a tumor of this type in a boy of 4.

From the Otolaryngological Department, Morrisania City Hospital.

1. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 321.

2. Ritchie, W. A., and Warren, S. L.: The Occurrence of Multiple Bony Lesions Suggesting Myeloma in the Skeleton of a Pre-Columbian Indian, Am. J. Roentgenol. **28**:622 (Nov.) 1932.

3. Atkinson, F. R. B.: Multiple Myeloma, M. Press **195**:312-317 and 327-333 (Oct. 13) 1937.

4. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 2, New York, American Journal of Cancer, 1936, p. 441.

5. Batts, M., Jr.: Multiple Myeloma: Review of Forty Cases, Arch. Surg. **39**:807 (Nov.) 1939.

6. Coley, W. B.: Multiple Myeloma, Ann. Surg. **93**:77 (Jan.) 1931.

7. Slavens, J. J.: Multiple Myeloma in a Child, Am. J. Dis. Child. **47**:821 (April) 1934.

The predominance of males suffering from the disease is striking. The ratio of males to females affected is 2 to 1.

The cause is obscured in a manner characterizing that of all malignant disease.

According to Ewing,[1] multiple myeloma is a specific malignant tumor of the bone marrow, probably arising from a single cell type and characterized mainly by multiple foci of origin. MacCallum [8] stated the opinion that the simultaneous appearance in many bones of numerous lesions points to a systemic involvement of the marrow cells.

The malady does not exist solely as diffuse myelomatosis. Extramedullary tumors, classified as plasmacytoma, are found chiefly in the nasopharynx, alveolar borders, tongue, lip and cervical lymph glands as solitary isolated growths. Beck [9] said: "The occurrence of myeloma in the field of otorhinolaryngology or even the borderlines of the same is very scarce." In 1931 Claiborn and Ferris [10] found 12 cases and added 2 of their own in which there was solitary involvement of the nose and nasopharynx. In 1936 Blumenfeld [11] raised this figure to 21. These authors cited 2 cases previously reported (Vogt,[12] 1912, and Wachter,[13] 1913) in which laryngeal involvement by a solitary tumor was discovered. These 2 cases and the one incorporated in this report are the only instances of attack on the larynx by myeloma, either primary or metastatic, noted in a diligent survey of the literature. A plasmacytoma involving the petrous pyramid, the mastoid process and the base of the skull was reported by Cappell and Mathers.[14] Hauser and Brownell [15] mentioned a patient having a rectal lesion which was proved to be myeloma of the plasmacytoma type, with a similar mass at the pharyngeal ostium of the left auditory tube. More recently Roh [16] described a case

8. MacCallum, W. G.: A Text Book of Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1937, p. 886.

9. Beck, J. C.: Some Uncommon Types of Neoplasms About the Head (Multiple Myeloma), Laryngoscope **40**:516 (July) 1930.

10. Claiborn, L. N., and Ferris, H. W.: Plasma Cell Tumors of the Nasal and Nasopharyngeal Sinuses, Arch. Surg. **23**:477 (Sept.) 1931.

11. Blumenfeld, L.: Plasma Cell Tumors of the Nose and Nasopharynx, Ann. Otol., Rhin. & Laryng. **45**:436 (June) 1936.

12. Vogt, E.: Granulomatosis plasmacellularis colli, Frankfurt. Ztschr. f. Path. **10**:129, 1912; cited by Blumenfeld [11] and Claiborn and Ferris.[10]

13. Wachter, H.: Ein Fall von multiplem Plasmazytom der oberen Luftwege. Arch. f. Laryng. u. Rhin. **28**:69, 1913; cited by Blumenfeld [11] and by Claiborn and Ferris.[10]

14. Cappell, D. F., and Mathers, R. P.: Plasmacytoma of the Petrous Temporal Bone and Base of the Skull, J. Laryng. & Otol. **50**:340 (May) 1935.

15. Hauser, I. J., and Brownell, D. H.: Malignant Neoplasms of the Nasopharynx, J. A. M. A. **111**:2467 (Dec. 31) 1938.

16. Roh, R. F.: Primary Mixed Cell Myeloma of the Mouth Involving the Antrum, Arch. Otolaryng. **31**:669 (April) 1940.

in which the maxillary antrum was involved from the alveolar border. In addition to involvement of these localities of interest to the otolaryngologist, Ewing [1] noted that extramedullary growths have been observed in many other regions of the body.

When multiple foci of origin are absent and a solitary myeloma of bone is observed, according to Pasternack and Waugh,[17] the entity offers a good prognosis. A greater weight of authoritative opinion holds that the solitary tumor is the initial lesion and that with the passage of time multiple involvement will be observed. King [18] offered a case report in support of this theory.

The first diagnosis in a case of multiple myeloma is generally wrong. The insidious nature of the onset is a constant characteristic. Geschickter [19] advanced six cardinal diagnostic features of this disease.

1. Multiple involvement of the skeletal trunk by tumor formation in an adult over 35 years of age. These neoplasms confine themselves for the most part to the sites of the red bone marrow. The skull, ribs, sternum, vertebrae, pelvic bones and femur are attacked in the order given. Ewing [1] observed twenty-eight areas of osseous destruction in the frontal and upper parietal area of one skull. The locale of these tumor formations is extremely painful.

2. Pathologic fracture of a rib. This accident has resulted from the simple exertion of turning over in bed.

3. Bence Jones bodies in the urine. Their presence is neither pathognomonic nor constant, proteinuria being noted in other diseases in which there is destruction of bone. The test for Bence Jones protein may serve as an index to the amount of destruction present.

4. Lumbar backache with signs of early paraplegia. Wandering intermittent mild rheumatic pains are among the first symptoms of myeloma.

5. An otherwise inexplicable anemia. This is a late sign and results from the destructive action of multiple tumors on the blood-forming marrow. The sedimentation rate is elevated, on account of the rapid formation of rouleaux, and sudden clumping adds difficulty to the accuracy of a red cell determination.[20] Cross matching and typing of

17. Pasternack, J. G., and Waugh, R. L.: Solitary Myeloma of Bone: A Clinical Entity, Ann. Surg. **110**:427 (Sept.) 1939.

18. King, B. B.: Solitary Plasma Cell Myeloma of Bone as an Initial Stage of Multiple Myeloma, J. A. M. A. **115**:36 (July 6) 1940.

19. Geschickter, C. F., and Copeland, M. M.: Multiple Myeloma, Arch. Surg. **16**:807 (April) 1928. Geschickter, C. F.: Multiple Myeloma as a Single Lesion, Ann. Surg. **92**:425 (Sept.) 1930.

20. Eklund, C. M.: Multiple Myeloma, Univ. Minnesota Hosp. **7**:142 (Jan. 30) 1936.

the blood may be impossible. If necessary, intravenous administration of blood serum or Ringer's solution should be resorted to.[21]

6. Chronic nephritis with retention of nitrogen, and low blood pressure. A satisfactory explanation of this and of the Bence Jones proteinuria has never been given. Low blood pressure accompanies cachexia.

Roentgen examination of the skull and long bones, microscopic study of marrow obtained by sternal puncture and biopsy of an accessible tumor will settle the diagnosis. The roentgen findings are characteristic. The multiple lesions appear as clearcut punched-out areas of decreased density, with no evidence of bone production or healing.[21] The normal bone marrow is absorbed and replaced by malignant plasma cells.

Sternal puncture as a diagnostic aid is mentioned once in the English literature. Haden and Rumsey [22] expressed the belief that an absolute diagnosis can be made by roentgenograms and a demonstration of the preponderance of plasma cells in the marrow. A specimen is obtained from the sternum at the level of the third interspace by means of a small drill devised by Favorite.[23] Staining with Wright's stain is satisfactory. More frequent use of this aid to diagnosis should be adopted.

In the gross myeloma are spherical soft elastic friable masses, gray or reddish, depending on their degree of vascularity. In size they vary from a few millimeters to 8 cm. in diameter.

Histologically the solitary tumors are identical with the multiple growths. They consist of loosely packed round or oval cells of different sizes, exhibiting features characteristic of plasma cells. The nuclei are single or multiple and are eccentrically placed. Mitotic figures are frequently seen and may have some relationship to the multiple nuclei of the larger cells. A constant feature of the nuclear chromatin is its deep-staining characteristic and its cartwheel or clock-faced arrangement.

Myelomas are classified into four types,[24] namely, plasmacytoma, lymphocytoma, myelocytoma and erythroblastoma. The classification of myeloma depends on the predominating cell or its possible site of origin. Ewing [1] stated the belief that these apparent variations may be due to

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21. Carlson, G. D.: Multiple Myeloma, Texas State J. Med. **31**:627 (Feb.) 1936.

22. Haden, R. L., and Rumsey, J. M.: Multiple Myeloma or Myelomatosis, M. Clin. North America **24**:369 (March) 1940.

23. Favorite, G. O.: An Instrument for Obtaining Bone Marrow, J. Lab. & Clin. Med. **25**:199 (Nov.) 1939.

24. (*a*) Ewing, J.: A Review of the Classification of Bone Tumors, Surg., Gynec. & Obst. **68**:971 (May) 1939. (*b*) Gross, R. E., and Vaughan, W. W.: Plasma Cell Myeloma: Report of Two Cases with Unusual Survivals of Six and Ten Years, Am. J. Roentgenol. **39**:344 (March) 1938.

different degrees of anaplasia in a single cell. Exclusive of plasmacytoma, these forms occur rarely and therefore receive scant attention in the literature.

Metastases in multiple myeloma are exceptional and more or less accidental. Myeloma cells do not enter the circulation as universally as do the pathologic cells of leukemia. Some authors, notably New and Harper,[25] have taken the position that the extramedullary growths are not true metastases but evidence of a systemic disease of the hemopoietic system. The finding of free tumor cells in the blood vessels of the bone marrow growths,[26] in the sinusoids of the liver and in the capillaries of the kidneys [27] seems to point conclusively to metastases by way of the blood stream.

The favorite sites for metastasis are the spleen, the liver and the lymphatic glands—all members of the hemopoietic system. Metastases have also been reported in the tonsil, nasopharynx, nasal accessory sinuses, ovaries, stomach, adrenals, muscular tissue and buccal cavity. In general, pulmonary metastases do not ocur. An extramedullary growth, which can be considered a metastasis, in the larynx of a patient with multiple myeloma is reported here. This anomaly has not been previously recorded in the literature.

The prognosis in multiple myeloma is always fatal. In 2 cases reported by Gross and Vaughan [24b] 1 patient lived ten years and the other six. Batts [5] pointed to the longest-lived patient on record—one who survived fourteen years and four months. The average time limit for this disease is two years.

Therapy is of no avail. Surgical removal or high voltage roentgen therapy to the solitary or multiple lesions serves only to prolong life a few months at the most.

REPORT OF A CASE

J. McK., a white man aged 69, was admitted to Morrisania City Hospital on Feb. 9, 1940. He complained of difficulty in breathing, pain on swallowing and a progressive loss of 32 pounds (14.5 Kg.) in weight.

The family history was unimportant.

Past History.—The patient was a bartender by trade and for many years had consumed whisky in large quantities. In 1937 a prostatectomy was performed, and in 1938 he was readmitted to the hospital on account of lumbar backache and pains in his shoulders. Roentgenograms of the spine and pelvis revealed hypertrophic changes, and numerous small areas of decreased density were observed in the frontal area of the skull at this time. These findings received scant attention,

25. New, G. B., and Harper, F. R.: Plasma Cell Myeloma of the Pharynx and Cervical Region Without Skeletal Involvement, Arch. Otolaryng. **16**:50 (July) 1932.

26. Geschickter and Copeland.[4] MacCallum.[8]

27. Scott, E.; Stanton, F. M., and Oliver, M.: Multiple Myeloma, Am. J. Cancer **17**:682 (March) 1933.

and symptomatic treatment was administered for the relief of pain. While he was in the hospital, "asthma" developed, which was unimproved on his discharge two months later.

Present Illness.—For the past nine months the patient had had increasing difficulty in breathing, which became distressing after mild exertion. In the past two months he had noticed an impediment on swallowing solid food. Deglutition was accompanied by a sticking pain in the region of the suprasternal notch.

Physical Examination.—The patient was an emaciated elderly white man, whose respiration was noisy and asthmatoid. External examination of the head showed nothing abnormal. The nose, mouth and pharynx were normal. The eyes showed arcus senilis. Slight tenderness was elicited over the larynx at the level of the cricoid cartilage. There was no cervical adenopathy.

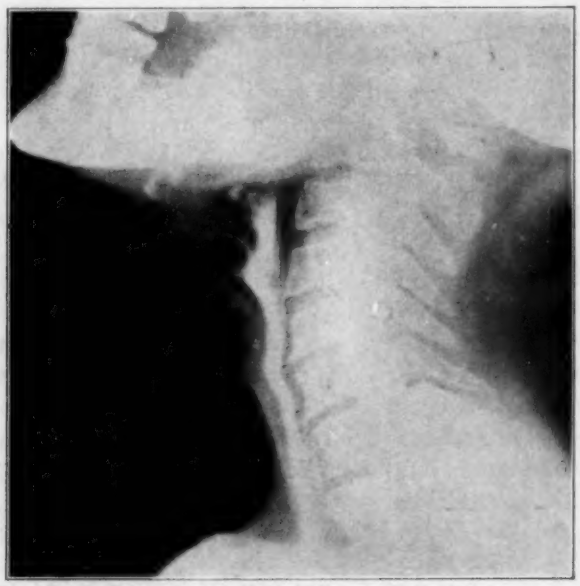

Fig. 1.—Myeloma nodule attached to the anterior surface of the signet of the cricoid cartilage.

Dry inspiratory and expiratory rales were so loud that the heart sounds were inaudible. The abdomen and extremities were normal.

Inspection by means of a mirror laryngoscope revealed a smooth round subglottic mass situated at the level of the cricoid cartilage. The lumen of the air passage was narrowed at this point and respiration embarrassed. A lateral cervical roentgenogram (fig. 1) showed an obstruction in the larynx at the level of the fifth cervical vertebra. Backward pressure of this mass presumably impinged on the esophagus during the act of swallowing.

Laboratory Data.—The red cell count was 3,000,000, and the white cells numbered 7,500. No abnormal forms were noted in the differential count. The Kahn reaction was negative. The values for serum calcium and serum phosphatase were normal. Repeated examinations of the urine did not reveal the presence of Bence Jones protein.

Roentgenograms demonstrated moderately well advanced productive arthritic changes in the lower lumbar vertebrae and old united fractures of the left fourth, fifth and ninth ribs in the postaxillary line. Throughout the vault of the skull numerous sharply defined circumscribed areas of bone destruction were present (fig. 2). There were also suggestive areas of lesser density in the mandible.

An attempt was made to secure a piece of the laryngeal mass by direct laryngoscopic procedure, but the tissue removed proved unsatisfactory for histologic examination.

Nine days after admission the dyspnea increased and a tracheotomy was performed.

Fig. 2.—Roentgenogram of the skull, showing the sharply defined defects of multiple myeloma throughout the vault.

One week subsequent to this operation, bleeding from the tracheotomy tube began. This was persistent and profuse. My associates and I decided that it originated from the laryngeal neoplasm and considered an exploratory laryngofissure necessary to cope with the emergency. This was done with the region under local anesthesia, in the usual manner except that the cricoid and the cricothyroid membrane were severed together with the thyroid cartilage, to secure a satisfactory exposure of the tumor mass. This was smooth, rounded and covered with intact thickened mucous membrane at all but one point, where there was surface ulceration. Active bleeding was present in this region. The tumor was a soft gray gelatinous mass having the consistency of sponge rubber. This was removed with a large mastoid curet. Its base rested on the anterior surface of the signet of the cricoid, and this cartilaginous barrier was intact.

Histologic examination of the removed tissue showed a growth of large plasma cells in a loose stroma. The nuclei were eccentrically placed and highly chromatic. Cartwheel arrangements and mitotic figures were frequently observed. The cytoplasm was acidophilic (fig. 3).

Marrow obtained by sternal puncture was characterized by an abnormal proliferation of plasma cells.

The patient remained in the hospital several weeks, received high voltage roentgen therapy and was discharged improved. He still wears the tracheotomy tube, which is corked the greater part of the time.

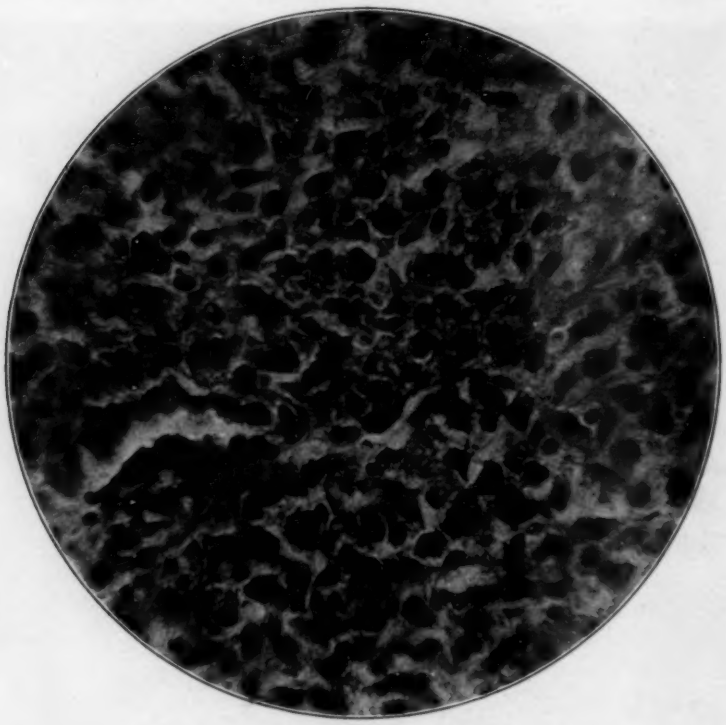

Fig. 3.—Photomicrograph of laryngeal nodule.

SUMMARY

1. Multiple myeloma is a relatively rare disease, which occasionally invades the areas that are treated by otolaryngologists. It manifests itself as a solitary extramedullary tumor or as a part of a general myelomatosis. Metastases in multiple myeloma are rare.

2. A case of multiple myeloma with a metastasis in the larynx is reported. Solitary involvement in this locality has been recorded twice previously, but a record of a metastatic growth has not been found.

Dr. Clarence H. Smith contributed suggestions for the preparation of this article.

2940 Grand Concourse.

THE LEMPERT ENDAURAL, ANTAURICULAR SURGICAL APPROACH TO THE TEMPORAL BONE

SUGGESTED MODIFICATION OF THE TECHNIC IN TYMPANO-MASTOIDECTOMY; REPORT OF FIFTY-FIVE CASES

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HILLERØD, DENMARK

Any operation should follow the route affording the best view of the surgical field, and that route should be as short as is compatible with due consideration for healthy tissue. The postaural incision is the direct approach to the mastoid process, but it gives a limited view of the middle ear. The approach through the external auditory canal leads directly to the tympanic cavity, but with the previously available methods it is practicable by this route to resect the cell system of the mastoid process only to a limited extent; besides, the field of vision is small, and the room for operative maneuvers limited.

The Lempert endaural approach to the temporal bone offers the great advantage that the surgeon is enabled at once to resect any cell system, regardless of its extent, just as through the postaural incision, and at the same time to have a direct view of and access to the tympanic cavity. Moreover, this operation gives an extraordinarily fine result cosmetically.

The technic has been described in detail by Lempert,[1] by Babbitt [2] and, for Scandinavian readers, by Kettel.[3] By means of three endaural, antauricular extracartilaginous incisions, varying in their course with the aim of the operation—tympanomastoidectomy (fig. 1 *A*) or simple mastoidectomy (fig. 1 *B*)—a triangular flap of the posterior membranous wall is circumcised and extirpated. The auricle, with all its attachments, is separated subperiosteally from the outer cortex of the mastoid and the zygoma, and, sharp retractors having been introduced, a mobile endaural membranous extracartilaginous window is created, which exposes to view any part of the mastoid where surgical measures are desired.

From the Fredericksborg County Hospital.

1. Lempert, J.: Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. **27**:555 (May) 1938.

2. Babbitt, J. A.: Endaural Surgery of Chronic Suppurations, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, p. 234.

3. Kettel, K.: Lempert's Endaural Approach to the Temporal Bone, Nord. med. (Hospitalstid.) **5**:377, 1940.

Having studied Lempert's endaural technic in his clinic in the spring of 1939, I introduced this excellent operation in my ward of the Fredericksborg County Hospital, where 35 tympanomastoidectomies, 2 modified tympanomastoidectomies and 18 simple mastoidectomies have since been performed after the method of Lempert. On the basis of my results I shall try to characterize the value of the endaural approach.

THE ENDAURAL APPROACH IN TYMPANOMASTOIDECTOMY

Tympanomastoidectomy is the principal indication for the use of the Lempert method, which in this particular field offers more advantages than does any other method. The Lempert endaural approach differs

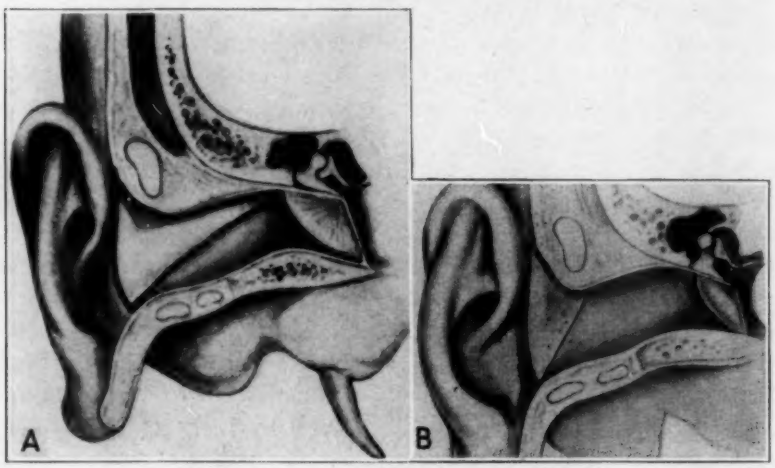

Fig. 1 (Lempert [1]).—*A*, technic for tympanomastoidectomy. A triangular flap of the posterior membranous wall is circumcised and extirpated. *B*, technic for simple mastoidectomy. Position of the endaural incisions.

from the postaural in giving an ideal direct and complete view of the tympanic cavity (fig. 2)—not a limited one from an oblique angle. This is the feature that more than anything else makes it so valuable. Furthermore, the use of this approach precludes the treatment of a postauricular wound and insures a beautiful result cosmetically. A direct view of the tympanic cavity is obtained also in the Thies–von Eicken endaural tympanomastoidectomy, it is true, but the visual angle is much smaller and the room for operative maneuvers far more limited; besides, because of the narrow space the operation can be performed only if the mastoid process is eburnated or if at any rate pneumatization is greatly

reduced, and finally, the operation is contraindicated in cases in which intracranial complications are suspected (Lund [4]). With the Lempert endaural approach the anatomy of the cell system is of no importance, and the value of this method is most obvious in cases in which tympanomastoidectomy is merely the first step in an operation on the labyrinth or the apex.

Henner [5] reported that he had performed 2 modified tympanomastoidectomies and 20 tympanomastoidectomies by this approach, while Woodruff [6] reported 40 and Howarth and Bateman [7] 10 tympanomastoidectomies. They all stated that they were greatly impressed by

Fig. 2.—Photograph taken during an operation for chronic suppurative otitis media on a patient with marked fistula symptom. Note the excellent view of the promontory, the prominentia canalis facialis and the fistula on the lateral semicircular canal (just above the posterior part of the prominentia canalis facialis).

4. Lund, R.: The Conservative Radical Operation on the Middle Ear and the Mastoid Process Carried Out Endaurally Without Incision of the External Ear, According to the Method of the Thies Brothers, Dansk otolaryng. Selsk. forhandl., 1936-1937, p. 21.

5. Henner, R.: Experiences with Endaural Complete Mastoidectomy and Atticomastoidectomy, Arch. Otolaryng. **31**:1022 (June) 1940.

6. Woodruff, G.: Observations on the Endaural Mastoidotympanectomy of Lempert, Arch. Otolaryng. **31**:1024 (June) 1940.

7. Howarth, W., and Bateman, G. H.: The Endaural Approach to the Mastoid, Lancet **2**:1168, 1938.

the value of the Lempert method. Shambaugh Jr.[8] and Galloway [9] made the same statement. Lempert [1] himself had performed about 150 of these operations (1938).

A different view was presented by Wishart.[10] He expressed a preference for the postaural route not only for simple mastoidectomies but also for most "radical mastoidectomies which require the removal of much diseased tissue." He admitted that the endaural method gives a better view of the tympanic cavity than does the postaural method, but with the endaural approach he has been inconvenienced by hemorrhage, and he expressed the opinion that this method requires particular training and takes a longer time even in the hands of an expert.

Like Babbitt,[11] I am unable to subscribe to these technical objections. On the contrary, to me the operation appears to proceed more rapidly and safely, because the view of the operative field is far clearer and more comprehensive with this method.

In one respect—and one only—has the Lempert operation caused me some difficulty. Lempert sacrifices the membranous covering of the entire posterior wall of the external auditory canal. Consequently, the epithelization of the cavity has to take place through ingrowth of epithelium from the floor and roof of the external auditory canal, as well as from the line of incision along the anterior border of the concha (fig. 1). This takes some time, however, and in 3 cases I was not able to prevent the formation of granulations until the facial ridge had been covered by epithelium, so that inconvenient adhesions formed; besides a stricture of the external auditory canal developed. Woodruff [6] reported a similar experience. In 2 of my cases it became necessary to revise—also endaurally—the cavity and to cover it with Thiersch grafts, after which the wound healed satisfactorily.

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8. Shambaugh, G. E., Jr., in discussion on Lempert, J.: Improvement of Hearing in Otosclerosis: Report of Seventy-Three Cases, Arch. Otolaryng. **30**: 144 (July) 1939.

9. Galloway, T. C., in discussion on Wishart [10] and in discussion on Woodruff.[6]

10. Wishart, D. E. S.: The Endaural Route, Arch. Otolaryng. **31**:218 (Jan.) 1940; Laryngoscope **49**:943, 1939. The reference to Dr. Wishart's point of view is based entirely on his paper, read to the American Otological Society May 23, 1939 and abstracted in the Archives of Otolaryngology (**31**:218 [Feb.] 1940). Not until the paper presented here was ready for print did I become aware of Wishart's original paper, in the Laryngoscope (**49**:943, 1939). It appears that he must have used a modification similar to that here suggested by me, as he wrote: "By a slight modification of the skin incisions and of the subsequent technique, the skin described as excised and thrown away can be used to line the mastoid bowl."

11. Babbitt, J. A., in discussion on Wishart.[10]

In order to avoid these two inconveniences I worked out a modification of the Lempert method, making a flap of the membranous covering of the entire posterior wall—a measure that facilitates the epithelization

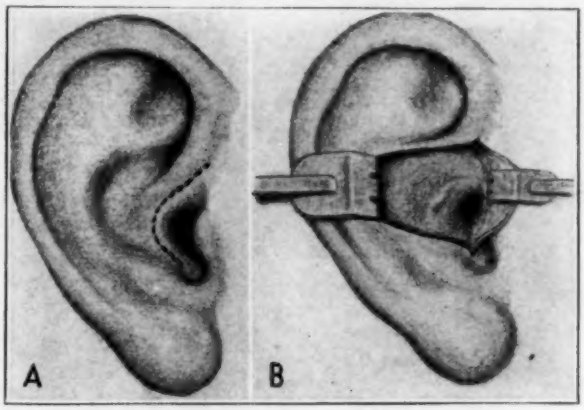

Fig. 3.—*A*, first step in the Lempert operation as modified by me: An arcuate incision is made, reaching from the apex of the trigonum suprameatum to the lower end of the anterior border of the concha and dividing the soft parts down to the bone. *B*, the endaural window has been created, and the entire membranous lining of the auditory canal is being pulled forward.

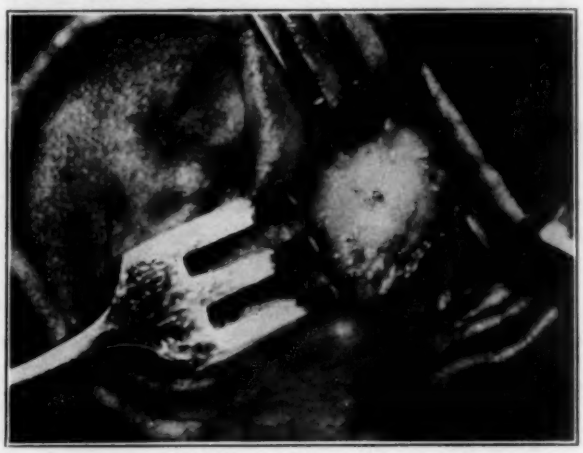

Fig. 4.—Photograph illustrating the same stage as fig. 3 *B*. Note the excellent exposure of the planum mastoideum. By shifting of the retractors the tip can be exposed.

to a great extent. Subsequently I have noted that Wishart,[10] also employing a method not described in detail, makes a satisfactory flap for the facial ridge and the mastoid bowl. According to the abstract

of his paper, however, Wishart [10] extirpates a triangle of the posterior membranous wall. But by omission of this step a far better flap is obtained. Woodruff [6] in 1 of his 40 cases saved the skin but questioned the advantage. This, judging from my experience, is great. The procedure employed by me is as follows:

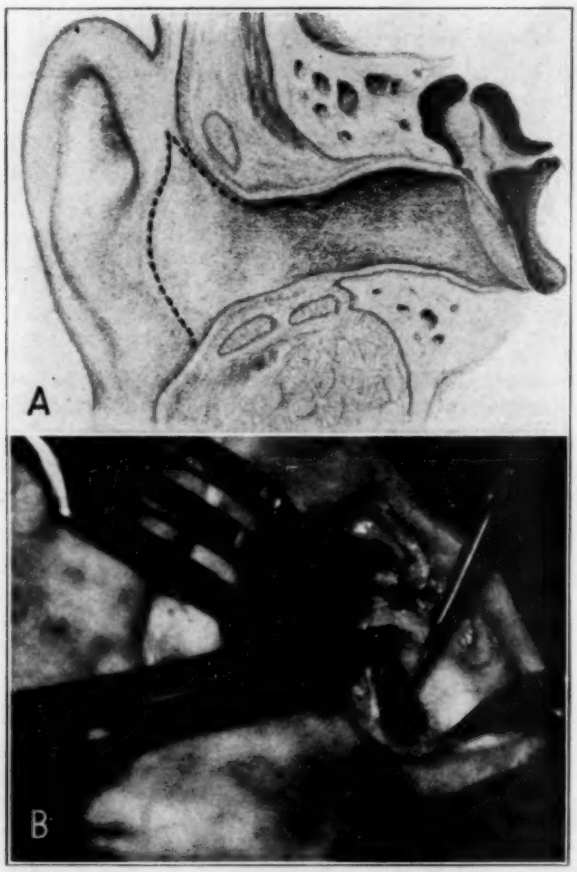

Fig. 5.—*A*, the second incision extends along the roof of the external auditory canal from the tympanic membrane to the apex of the trigonum suprameatum, where it joins the beginning of the first incision. *B*, photograph showing knife beginning the incision described under *A*.

MODIFICATION OF THE LEMPERT TECHNIC IN TYMPANOMASTOIDECTOMY

An incision is made reaching from the apex of the trigonum suprameatum to the lower end of the anterior border of the concha—i. e., identical with Lempert's third endaural incision (fig. 3*A*.). The auricle with its attachments is separated

from the outer cortex of the mastoid and the zygoma, and sharp retractors are introduced. At the same time the membranous covering of the entire posterior wall is separated from the underlying surface of the bone and pulled forward by means of a blunt retractor (figs. 3*B* and 4). Now the antrum is opened, and the posterior osseous wall is removed. The blunt retractor is removed from the membranous covering of the posterior wall, which is then incised along the roof

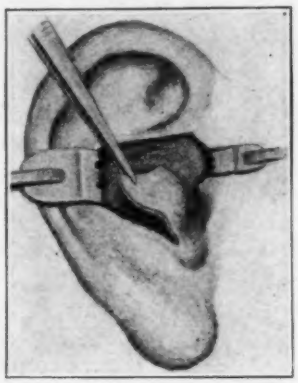

Fig. 6.—A flap has been created for covering the facial ridge and the mastoid bowl.

Fig. 7.—Photograph showing same stage as fig. 6.

(fig. 5). After the subcutaneous tissue of the flap has been partially removed, it is placed over the facial ridge and the mastoid bowl (figs. 6, 7, 8 and 9). The rest of the cavity is covered primarily with Thiersch grafts, any part of the sinus and dura incidentally exposed being left uncovered.

The advantage of the original Lempert technic in comparison with the modification here suggested is that the former allows a perfectly free

view of the tympanic cavity from the beginning of the operation. The drawback is that the epithelization of the cavity is protracted, making the after-treatment difficult. The suggested modification offers the

Fig. 8.—The flap has been placed so as to cover the facial ridge partially and line the mastoid bowl.

Fig. 9.—Photograph showing same stage as fig. 8. The prominentia canalis facialis and the lateral semicircular canal are seen, if a little dimly.

advantage of easier and more rapid incisions, besides that of more rapid epithelization of the cavity, stricture of the external canal being avoided. On the other hand, one does not get the same unhindered view of the tympanic cavity obtained by the original technic until the posterior osseous wall has been removed and the flap properly placed.

I have performed 35 tympanomastoidectomies and 2 modified tympanomastoidectomies in the way described, with a fatal outcome in 1 case.[12] In 5 of the tympanomastoidectomies I employed the original technic. Two of these operations had to be revised. In 32 cases (including a case of abscess in the temporal lobe of the brain) the modification was employed, and the result has been ideal, cosmetically as well as clinically.

Wishart[10] arrived at the conclusion that the endaural approach is not the method of choice for simple or radical mastoidectomy, and he cited the rules given by Whiting[13] for mastoidectomy, which call for "complete eradication of all diseased bone, in the shortest time commensurate with thoroughness, by methods designed to reduce to a

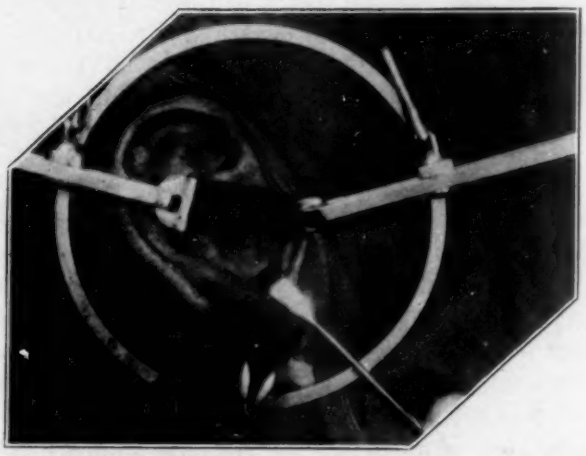

Fig 10.—Self-retaining retractor for the endaural technic, designed by B. H. Shuster (Mastoid Retractor, Self Retaining, for Endaural, Antauricular Technic, ARCH. OTOLARYNG. **30**:993 [Dec.] 1939). In my experience this instrument has given excellent service during the maneuvers in the middle ear, after resection of the mastoid cells.

minimum dangers incident to the operation and calculated as well to induce speedy and permanent healing."

As the endaural approach provides perfectly free access for the resection of any cell system, as the view of the tympanic cavity is ideal

12. A man of 40 years had an acute exacerbation of chronic otitis media, cholesteatoma with labyrinthitis, and acute purulent diffuse meningitis. His spinal fluid had the appearance of milk, and contained uncountable cells. Labyrinthectomy was performed endaurally, after the method of Neumann. He died four days later, after temporary improvement.

13. Whiting, F.: The Modern Mastoid Operation, Philadelphia, P. Blakiston's Son & Co., 1905.

and the performance of operative maneuvers consequently quicker and safer, and as the treatment of a postaural wound is entirely avoided, I venture from the rules laid down by Whiting to draw a conclusion the opposite of Wishart's. For tympanomastoidectomies, particularly for operations on the labyrinth and apex, the Lempert endaural approach modified as here suggested is the method of choice.

THE ENDAURAL APPROACH IN SIMPLE MASTOIDECTOMY

Indispensable conditions for the employment of the endaural route for simple mastoidectomy are that the view of the field allows the operation to be performed with complete safety and that drainage is free.

Lempert [1] reported that he had performed no less than 1,595 simple mastoidectomies endaurally and that it had been possible by the use of this route to treat any complication incident to the operation. Howarth and Bateman [7] reported that they had performed 20 simple mastoidectomies and that this operation is the crucial test for the value of the endaural approach, as it is impossible to know beforehand how far the pathologic processes extend in a given case. Their material includes instances of perisinal abscess as well as extradural abscess, but in all their cases the course was perfectly satisfactory. Henner [5] reported 16 operations of this kind, and he stated that by the endaural approach every type of pathologic condition of the mastoid can be dealt with adequately. He admitted, however, that "the only difficulty encountered has been that in a few of the cases in which the disease was in an early stage, particularly when there was edema of the skin, exposure of the tip was difficult." This is a point I wish to deal with more thoroughly.

It is to be kept in mind that the endaural approach is dependent on the formation of a mobile window, which "permits an adequate exposure of every part of the mastoid portion of the temporal bone, not because of the actual surgical field it directly exposes, but because of the field it can be made to expose as a result of its mobility" (Lempert [1]). The mobility is an explicit condition, however, and if it is lowered by swelling of the soft parts, a complete resection of the cells will present difficulties. This has been emphasized by Wishart [10] and by Galloway,[9] and it has been admitted by Henner.[5] From entirely theoretic considerations I have held the same view, and in every case in which swelling of the soft parts was involved I therefore have preferred the postaural route.

At first the drainage is perfectly free, but, as pointed out by Wishart [10] and emphasized especially by Babbitt,[11] inconvenient granulations may form in the endaural window, obstructing the free outflow. Consequently the after-treatment entails close watching by the surgeon.

On the other hand, though Wishart [10] claimed that he felt lucky because he had seen infection of the temporal fascia resulting from the use of the endaural approach in only 1 case, I agree with Babbitt [11] that this risk may be left out of consideration completely. In only 1 case out of 55 have I encountered an abscess, quite superficial, in the cicatrix, and this was entirely due to faulty technic, as I had overzealously sutured the small incision—which is against the directions given by Lempert.[1] After the suture was removed, the small abscess disappeared immediately.

I quite agree with the authors who claim that the exposure of the surgical field is excellent and that every cell system and also the systems located behind the sinus and down in the tip may be removed without difficulty. This was the case with 3 of my patients, 2 of whom had a perisinal abscess and 1 an extradural abscess. Yet these were dealt with most satisfactorily. I have had no cause to regret the use of the endaural approach. As pointed out by several authors, the technic of this operation, as of any other, has to be learned, but it is easily acquired.

Naturally, little may be deduced from only 18 cases, and consequently I prefer, like Henner [5] and Woodruff,[6] to reserve my judgment until I shall have had more experience. At present I hold it justifiable to employ the endaural approach for simple mastoidectomy in all cases in which swelling of the soft parts does not impair the mobility of the endaural window. But I insist that the surgeon himself shall direct the after-treatment and watch that the drainage is kept free.

The postaural incision is the direct route to the mastoid process. It offers a perfectly free view, and it insures free drainage, but at the sacrifice of tissues (skin, subcutis, periosteum, musculature) which often are not involved in the morbid process. In the endaural approach, injury to these tissues is avoided. As the sternocleidomastoid muscle is not divided, there is less postoperative pain; there is no postaural wound to be treated, and there can be no disfiguring scar, with resulting inconveniences (pain, fistula). Finally, the patient recovers his normal appearance—a point that is often underestimated by the physician but highly appreciated by the patient.

The present estimation of the Lempert [1] endaural approach, I think, may be summed up as follows: With one exception (Wishart [10]) all authors with experience in this field agree that for tympanomastoidectomy and modified tympanomastoidectomy the endaural approach is the method of choice. Its advantages over the postauricular route are great. For simple mastoidectomy many authors recommend the endaural approach in preference to the postaural route, but opinions differ, and more experience has to be gained before the indications can be established safely.

SUMMARY

A report is made of 55 operations—35 tympanomastoidectomies, 2 modified tympanomastoidectomies and 18 simple mastoidectomies—performed with the Lempert endaural approach.

A modification of the Lempert endaural approach, by which the entire posterior membranous wall is preserved as a flap for covering the facial ridge and the mastoid bowl, is suggested as the method of choice for tympanomastoidectomy and modified tympanomastoidectomy.

The employment of the endaural approach is warranted also in simple mastoidectomy in cases in which the mobility of the endaural window is not impaired by swelling of the soft parts.

Dr. H. Stockholm made the drawings and Mr. N. Lichtenberg the photographs, some technical details of which are: Reflex Korelle 6 by 6 cm. camera; telephoto Xenar lens; f/8; exposure, one-fourth second; distance, 40 inches (100 cm.); lighting, two lamps, 250 candle power each, and Agfa Iso-pan F. film.

PNEUMOCOCCUS TYPE III IN OTITIC INFECTIONS

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The clinical picture of Pneumococcus type III infection of the ear has been presented from time to time since the original description of Pneumococcus, by Schottmüller [1] in 1903. Kümmel,[2] in 1907, Neumann, Ruttin and Gohn,[3] in 1909, Albert,[4] in 1911, Alberti,[5] in 1913, and others have presented descriptions of a definite clinical entity ascribed to this organism.

The importance of this entity has been recognized, and all its concomitant features should be viewed with a certain degree of gravity. Early surgical intervention usually is followed by an uneventful convalescence. However, because of the extremely insidious nature of the infection, with the otoscopic picture often atypical, operation is frequently delayed, and when this is true, intracranial complications are relatively more common. It seems that this clinical entity often has been accepted as incidental, for few American investigators have described the condition. A series of foreign articles have stressed the genesis of "mucosis otitis." The most recent and complete observations were those reported by Maybaum and Druss [6] in 1939.

The present survey includes 307 mastoidectomies performed from June 1, 1935 to Jan. 1, 1940 in the services of Drs. G. M. Coates and M. S. Ersner at the Graduate Hospital of the University of Pennsylvania and Mount Sinai Hospital, respectively. In 195 cases the infection was acute, and in 112 it was chronic. Unfortunately, reports of cultures for the entire series are not available; positive cultures were reported in 154 of the cases in which infection was acute and in

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From the Otologic Service of Drs. G. M. Coates and M. S. Ersner at the Graduate Hospital of the University of Pennsylvania and Mount Sinai Hospital, respectively.

1. Schottmüller, H., cited by Maybaum and Druss.[6]

2. Kümmel, W.: Klinische und bakteriologische Beobachtungen über akute Mittelohreiterung, Ztschr. f. Ohrenh. **52**:177, 1906.

3. Neumann, Ruttin and Gohn, cited by Maybaum and Druss.[6]

4. Albert: Beitrag zur Bakteriologie der Otitis media purulenta akuta, Arch. f. Ohrenh. **85**:251, 1911.

5. Alberti, O.: Zur Bakteriologie der akuten Mastoiditis, Arch. f. Ohrenh. **92**:111, 1913.

6. Maybaum, J. L., and Druss, J. G.: Otitic Infections Due to Pneumococcus Type III, Arch. Otolaryng. **30**:21 (July) 1939.

only 36 of those in which it was chronic. This discrepancy may have been due to neglect in examining the culture material, to faults or delay in submitting this material to the laboratory or to lack of growth of organisms in the culture mediums. Thus, this survey involves 190 reported cultures. Table 1 shows the grouping of the various organisms. Pneumococcus was found in 33 of 190 cultures which were reported positive, an incidence of 17.3 per cent, or of 10.8 per cent of the 307 cases covered by the survey. This incidence is comparable to that reported by me [7] in an earlier survey (Pneumococcus in 16 of 162 positive cultures).

It also compares with the reports of Kümmel (11 per cent), Denker [8] (14 per cent), Albert (16.4 per cent), Hesse [9] (17.6 per cent), Page [10]

TABLE 1.—*Classification of Organisms Isolated from Mastoids*

			Cases
Streptococcus			118
	Hemolytic	96	
	Nonhemolytic	21	
	Viridans	1	
Staphylococcus			27
	Aureus Hemolytic	3	
	Nonhemolytic	1	
	Albus Hemolytic	13	
	Nonhemolytic	10	
Bacillus pyocyaneus			1
Pathogenic diphtheroids			2
Bacillus proteus			7
Bacillus influenzae			1
Pneumococcus			33
Unidentified cocci			1
Total			190

(10.4 per cent), Vaheri [11] (27 per cent)—rather high—and Eckert-Möbius [12] (12 per cent). My figures are based on a general grouping of the pneumococci. Table 3 shows that there were 26 cases of the type

7. Coates, G. M.; Ersner, M. S., and Persky, A. H.: A Study of Mastoiditis: A Résumé of Six Hundred and Forty-Four Cases, Laryngoscope **40**:245 (April) 1930.

8. Denker, A.: Ueber bakteriologische Untersuchungen bei akute Mittelohreiterung, Ztschr. f. Ohrenh. **54**:200, 1907.

9. Hesse, W.: Beiträge zur Bakteriologie und Klinik akuter Mittelohreiterungen, akuter Mastoiditiden und deren Komplikationen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:372, 1929.

10. Page, J. R.: Bacteriology of Acute Infections of the Middle Ear and Mastoid, Arch. Otolaryng. **20**:447 (Oct.) 1934.

11. Vaheri, E.: Bakteriologisch-klinische Untersuchungen über operativ behandelte Komplikationen der akuten Mittelohrentzündung, Acta oto-laryng., 1938, supp. 29, p. 1.

12. Eckert-Möbius, A.: Die Mukosusotitis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **116**:270, 1927.

III infection. While this limitation will lower all percentages, it does not appear to influence the general considerations presented in this survey.

There were 23 male and 10 female patients. The age incidence of pneumococcic aural infections is noted in table 4. While there was a tendency to an increase of such infections in the later decades of life, the greatest frequency was still observed in the first three decades. This finding is at some variance with that of Maybaum and Druss [6] and of Vaheri.[11] The latter found that 58 per cent of his patients were in the sixth decade. The age of my youngest patient was 8 months, while that of the oldest was 73 years.

Much has been written concerning the establishment of a pathologico-clinical entity which can be identified with pneumococcic infection. Because of the insidious, progressive nature of the infection and the frequent association of intracranial complications, many investigators have attempted to build up a typical clinical picture. Certain characteristics are noteworthy. The pathologic change is usually a slow progressive softening and necrosis of the mastoid cells and intracellular septums. This pathologic process can be anticipated more or less, since it has been shown in the numerous discussions on this subject that Pneumococcus seems to have a predilection for the well pneumatized mastoid process. Vaheri stressed this factor, and, acknowledging the impossibility of making a preoperative forecast, made a roentgenologic grouping according to the types of pneumatization found in his series of cases. He followed Barth's [13] classification of the types of pneumatization, with minor modifications, to wit:

1. Extensive pneumatization.
2. Good pneumatization, with large cells extending to the squamosa and the zygomatic root. These cells may be distributed either regularly or irregularly.
3. Pneumatization limited to the mastoid or to certain areas of the mastoid process.
4. Pneumatization limited to the antral region.
5. Compact mastoid.

In his review, he found that mastoids in the first two groups were invaded principally by Pneumococcus and definitely more frequently by this organism than by Streptococcus. My experience has been similar in this respect, as is seen in table 5. This shows that 16 mastoids presented good or extensive pneumatization. Most of these were in the older age group. It is unfortunate that complete roentgen reports

13. Barth, H., cited by Vaheri.[11]

TABLE 2.—*Data on Patients with Otitic Infection Due to Pneumococcus Type III*

Patient				Symptoms				Operative Results								
No.	Name	Sex	Age, Yr.	Cause	Duration	Temperature on Admission, F.	Physical Findings	Site, Right or Left	Roentgen Findings	Operative Procedure	Findings	Culture Type	Postoperative Stay in Hospital	Complications	Results	Comment
1	C. U.	F	67	Grip	4 weeks	102	Comatose; nuchal rigidity	L	None	Simple exposure of lateral sinus and dura	Extensive necrosis; exposed lateral sinus and posterior fossa	III	1 day	Meningitis	Death	Admitted with frank meningitis; spinal fluid 3,234 cells; Pneumococcus type III in fluid
2	J. R.	M	67	Acute infection of the upper respiratory tract	3 days	99.2	Irrational; nuchal rigidity	R	Sclerotic mastoid with necrosis	Simple exposure of dura and lateral sinus	Extensive necrosis; extradural and perisinal abscess	III	3 days	Meningitis	Death	Admitted with frank meningitis, diabetes, syphilis; spinal fluid 900 cells; Pneumococcus type III in fluid; simple mastoidectomy 5 weeks before time of writing; Pneumococcus type III recovered
3	A. S.	M	..	.....	3 days	103	Comatose; postauricular swelling	L	None	None	.....	I	1 day	Meningitis	Death	Admitted with frank meningitis; comatose; spinal fluid 1,100 cells; Pneumococcus type III in fluid
4	G. O.	M	4	.....	15 days	98	Postauricular swelling; discharge	L	None	Simple mastoidectomy	Subperiosteal abscess; necrosis	III	6 days	Subperiosteal abscess	Cure	Normal temperature; painless postauricular swelling for 10 days
5	A. F.	F	48	.....	10 days	99	Preauricular swelling	R	Good pneumatization	Simple mastoidectomy	Subperiosteal abscess in zygomatic region	III	7 days; 23 days	Subperiosteal abscess; osteomyelitis of temporal bone	Cure	Late complication of osteomyelitis; Staphylococcus albus haemolyticus
6	M. B.	M	49	Bathing	3 months	98	Postauricular swelling; discharge	L	Good pneumatization	Simple mastoidectomy	Perisinal abscess; necrosis	III	8 days	Perisinal abscess	Cure	Slow onset; multiple paracentesis; extensive pneumatization; perisinal abscess
7	M. M.	F	56	Cold	4 days	103	Comatose; nuchal rigidity	R	None	None	Extensive necrosis (autopsy)	XIV	3 days	Meningitis	Death	Admitted with frank meningitis; spinal fluid contained Pneumococcus type IV; history of acute infection of short duration

8	W. S.	M	22	Bathing	2 weeks	99.8	Discharge; postauricular pain	R	Extensive pneumatization and necrosis	1. Simple mastoidectomy 2. Radical mastoidectomy	Extensive necrosis; granulations	I	10 days	Meningitis	Death	Meningitis developed 2 days after admission; spinal fluid 7,000 cells; Pneumococcus type I in fluid
9	P. S.	M	29	.....	1 year	98.6	Facial palsy; discharge	R	Moderate pneumatization	Radical exposure of facial nerve	Cholesteatoma	...	1 month	Preoperative facial palsy	Cure	Slow long history of cholesteatoma; facial palsy; Pneumococcus (unidentified type)
10	S. K	F	37	Cold	1 month	98.6	Watery discharge; dizziness; unilateral headaches	L	Extensive pneumatization	Simple mastoidectomy	Extensive necrosis	III	9 days	None	Cure	
11	J. K.	M	29	.....	1 month	98.6	Watery discharge; postauricular pain	L	Diploic mastoid	Simple mastoidectomy	Thrombotic necrosis	III	8 days	None	Cure	Diploic mastoid
12	D. R.	M	3½	.....	6 weeks	101.4	Postauricular swelling; discharge	L	Moderate pneumatization	Simple mastoidectomy	Necrosis	III	12 days	None	Cure	
13	I. G.	M	57	.....	4 months	98.6	Postauricular pain; deafness	R	Mixed	Simple mastoidectomy	Sclerosis; extradural abscess; thrombosis of lateral sinus	III	7 days	Extradural (cerebellar) abscess; sinus thrombosis	Cure	Extensive necrosis in an extensive pneumatization; (silent mastoid) uneventful recovery
14	Same as no. 13, 2 months later					100	Deafness; postauricular pain; swelling	L	Good pneumatization	Simple mastoidectomy	Subperiosteal, von Bezold and zygomatic abscesses	III	13 days	Subperiosteal, von Bezold and zygomatic abscesses	Cure	Same as 13
15	V. M.	M	23	.....	3 weeks	98.6	Discharging ears; deafness	R and L	Mixed	Bilateral mastoidectomy	Extensive necrosis	III	8 days	None	Cure	
16	M. B.	F	15	.....	.....	103	Discharge; postauricular pain	L	Extensive pneumatization	Simple mastoidectomy	Hemorrhage and extensive necrosis	III	9 days	None	Cure	Temperature 103 F.; extensive necrosis
17	L. A.	F	9	.....	2 weeks	99.6	Postauricular pain; discharge	R	None	Simple mastoidectomy	Sclerosis; subperiosteal abscess; necrosis	III	9 days	Subperiosteal abscess	Cure	Subperiosteal abscess in a sclerotic mastoid
18	J. L.	M	60	Cold	2 months	98	Postauricular pain; discharge	R	None	Simple mastoidectomy	Subperiosteal, perisinal and extradural abscesses	III	11 days	Subperiosteal, perisinal and extradural abscesses	Cure	Intermittent discharge and earache for two months; extensive necrosis

TABLE 2.—*Data on Patients with Otitic Infection Due to Pneumococcus Type III—Continued*

Patient				Symptoms				Operative Results								
No.	Name	Sex	Age, Yr.	Cause	Duration	Temperature on Admission, F.	Physical Findings	Site, Right or Left	Roentgen Findings	Operative Procedure	Findings	Culture Type	Postoperative Stay in Hospital	Complications	Results	Comment
19	R. B.	F	4	.....	3 weeks	100.6	Postauricular pain; discharge	R and L	Good; pneumatization	Bilateral mastoidectomy	Extensive necrosis	III	8 days	None	Cure	
20	S. M.	F	12	.....	10 years	100.2	Internal discharge; pain	R	None	Simple mastoidectomy	Diploic mastoid	III	8 days	None	Cure	Diploic type of mastoid
21	B. S.	M	2½	Bronchial pneumonia	2 months	100	Discharge	L	Necrosis; good pneumatization	Simple mastoidectomy	Extensive necrosis	III	7 days	None	Cure	Bilateral discharge of 2 mo. duration, associated with bronchial pneumonia; uneventful recovery
22	M. F.	M	50	.....	2 weeks	97	Discharge; postauricular pain; tinnitus	L	None	Simple mastoidectomy	Extensive necrosis; perisinal abscess	III	2 weeks	Perisinal abscess	Cure	Intermittent discharge of 7 weeks' duration; mild labyrinthine symptoms immediately after operation; cleared up in 2 weeks
23	H. L.	M	38	Cold	3 weeks	99	Postauricular pain; discharge	L	Mixed	Simple mastoidectomy	Mixed cell necrosis	I	6 days	None	Cure	Intense hemicrania
24	C. C.	M	42	Cold	3 weeks	98	Postauricular pain; discharge	L	Extensive pneumatization	Simple mastoidectomy	Extensive necrosis; extradural perisinal and von Bezold abscesses	III	11 days	Extradural, perisinal and von Bezold abscesses	Cure	Extensive necrosis
25	B. C.	M	59	Grip	2 months	98	Postauricular pain; discharge	L	Sclerotic mastoid	Simple mastoidectomy	Extensive necrosis; perisinal abscess	III	6 days	Perisinal abscess	Cure	Sclerotic mastoid; intermittent discharge of 2 months' duration

26	R. L.	F	1½	Cold	1 week	104	Postauricular pain; discharge	R and L	Extensive pneumatization; necrosis	Bilateral mastoidectomy	Extensive necrosis	III	1 month	None	Cure	
27	W. F.	M	13	Cold	2 weeks	100	Postauricular pain; discharge	R	None	Simple mastoidectomy	Moderate necrosis	IV	12 days	None	Cure	Pneumococcus type IV
28	F. S.	F	7	Grip	5 weeks	100	Postauricular pain; discharge	R	None	Simple mastoidectomy	External necrosis	III	8 days	None	Cure	Intermittent discharge for 5 weeks; low grade fever
29	E. S.	M	11 mo.	Grip	2 weeks	101	Postauricular pain; swelling; discharge	L	Good pneumatization	Simple mastoidectomy	Coalescent necrosis	II	2 weeks	None	Cure	Secondary anemia; Pneumococcus type II
30	M. B.	M	8 mo.	Cold	3 weeks	98.8	Postauricular swelling	L	None	Simple mastoidectomy	Diploic mastoid and necrosis	III	7 days	Subperiosteal abscess	Cure	Postauricular swelling; no discharge; intact drum membrane (primary mastoid?)
31	P. R.	M	62	Grip	4 weeks	98.8	Headache; discharge	L	Good pneumatization	Simple mastoidectomy	Extensive necrosis; extradural and perisinal abscess	III	1 month	Extradural and perisinal abscess	Cure	Marked hemicrania; postoperative subacromial bursitis (suppurative)
32	P. C.	M	73	Cold	10 weeks	98.3	Hemicrania; discharge	R	Extensive pneumatization; necrosis	Simple mastoidectomy	Extensive necrosis; extradural and perisinal abscess	III	7 days	Extradural and perisinal abscess	Cure	Mastoiditis following eustachian catheterization; atypical symptoms; severe hemicrania; normal temperature
33	Same as no. 32, 3 weeks later					98.6	Same	L	Extensive pneumatization; necrosis	Simple mastoidectomy	Same as 32	III	5 days	Same as 32	Cure	Three weeks later, spontaneous perforation of the left ear; hemicrania; operative findings and convalescence identical with those of no. 32

were not available in all cases. Such reports would not only enhance the value of this study but would also make possible many interesting correlations. However, of the 11 patients for whom data on roentgen examinations are not available, 3 had meningitis. Operations on 7

TABLE 3.—*Classification of the Pneumococci Recovered from Material Taken from Mastoids*

Type	Cases
I	3
II	1
III	26
IV	1
XIV	1
Unclassified	1
Total	33

TABLE 4.—*Age and Sex Distribution of Patients*

Years	Patients
1 to 10	9
10 to 20	3
20 to 30	4
30 to 40	2
40 to 50	3
50 to 60	5
60 and over	6
Unknown	1
Total	33

Youngest patient	8 mo.
Oldest patient	73 yr.
Males	23
Females	10
Total	33

TABLE 5.—*Roentgen Classification of Mastoids According to Type of Pneumatization*

	Cases
Pneumatic	16
Mixed	3
Diploic	1
Sclerotic	2
No description reported	11
Total	33

additional patients revealed marked pneumatization of the mastoids with extensive necrosis.

Clinically, pneumococcic infection may follow a number of different courses. Maybaum and Druss [6] noted: (1) a prolonged clinical course with few or no physical signs, followed by evidence of mastoiditis or intracranial complication; (2) the usual clinical course of middle ear

infection, followed by mastoiditis or intracranial complication, and (3) a short clinical course (two to five days), followed by intracranial complication. The same conditions were observed in the patients of my series, as seen in table 6, which gives the duration of symptoms prior to admission to the hospital. The 3 patients with symptoms of less than one week's duration had meningitis and died shortly after admission. There were 17 in the second group, of whom 2 had meningitis, 6 had subperiosteal abscess, 6 had perisinal abscess and 13 had extensive necrosis. In the third group there were 8 with extensive necrosis, which included 4 with perisinal or epidural abscess, 1 with meningitis and 1 with thrombosis of the lateral sinus.

The symptoms indicate a slow progressive otitic infection. There is a persistent discharge, variable in character which is usually thin and watery but may be mucoid or mucopurulent. Though it may be profuse,

TABLE 6.—*Duration of Symptoms (Preoperatively) with Operative Findings*

Duration of Symptoms	Patients	Operative Finding	Patients
Less than 1 week	3	Meningitis	2
1 to 2 weeks	7	Extensive necrosis	13
2 to 4 weeks	10	Perisinal abscess	6
		Subperiosteal abscess	6
		Meningitis	2
1 to 2 mo.	8	Necrosis	8
Over 2 mo.	4	Perisinal abscess	4
		Cholesteatoma	1
		Meningitis	1
		Sinus thrombosis	1
Unknown	1		
Total	33		

it usually is only moderate in amount, and more or less constant. There does not seem to be any appreciable variation in quantity, and this invites a dangerous "watchful waiting" attitude. Postauricular pain is frequent, but it does not have the intensity of that experienced in the more virulent infections caused by the streptococci. On the other hand, there may be a dull persistent deep-seated pain, which may extend as far back as the occiput and upward to the vertex. It is a true hemicrania, in certain cases hard to describe and often difficult to associate clearly with the few otoscopic findings. However, when present it should be viewed with considerable apprehension, as it may signify some intracranial extension. Postauricular swelling is usually more difficult to elicit than in streptococcic infections. When it does occur, it appears rather suddenly and not as a result of periostitis but rather as a result of a perforation of the cortex (subperiosteal abscess or von Bezold's abscess).

Impaired hearing and tinnitus are frequently noted. The deafness may be marked and rather surprising in the presence of a drum

membrane that does not appear severely involved or destroyed. Fever and other evidences of toxemia are not dependable signs. Most of the patients in this series had a normal or only slightly elevated temperature. Excluding those with intracranial complications, only 6 were admitted with a temperature over 100 F.

The otoscopic picture is rather disconcerting. The drum membrane may be dull, lusterless, at times thickened, with the landmarks frequently present. As a rule the canal is not appreciably narrowed, although there may be a fulness in the superior-posterior half of the tympanum. The perforation is usually small and yet permits free egress of the thin discharge. Altogether, there does not seem to be any evidence of retention of pus under pressure.

From the laboratory standpoint there are mild leukocytosis and mild anemia, which become slowly more marked but assume more rapid and profound changes with the onset of any intracranial complication. The roentgen findings are significant, since (1) an extensive pneumatization

TABLE 7.—*Types of Mastoidectomy*

Type	Cases	Mastoids Involved			
		Right	Left	Both	Total
Simple (3 bilateral)	29				
Radical (1 meningitis; 1 facial palsy)	2				
None (2 meningitis)	2				
Total	33	12	18	3	33

of the mastoid process should be viewed seriously and (2) the presence of extensive destruction is readily demonstrated and hence will direct the otologist to early operative intervention in spite of any paucity of local clinical findings.

From the foregoing observations it is obvious that it is often difficult to appraise accurately the true significance of many of the symptoms and findings. It is also a problem to determine the optimum time to operate. The best surgical judgment is often biased because of the paucity of clinical findings and the atypical picture presented. Table 7 gives the various operative procedures carried out on the patients of my group. In spite of the fact that there were 29 simple mastoidectomies, there was a large proportion of complications, as is evidenced by table 8. These complications can be associated readily with extensive necrosis of the mastoid process. This later operative finding was reported in 20 of the cases. However, in spite of the frequency of extensive necrosis, with its associated complications, the period of convalescence did not seem to be influenced, for the usual stay in the hospital was relatively short

and usually uneventful. Secondary complications did not occur, as occasionally happens in the streptococcic infections.

Having viewed this problem from many angles, it seems that an index for the optimum time for operation can be established. In the presence of an otitic infection, slow in progress, somewhat atypical in course, with or without a positive culture of the discharge from the middle ear and with extensive pneumatization of the mastoid process (seen roentgenologically), it is well to consider early intervention. Although one may supplement the usual routine of treatment with the newer chemotherapeutic and serotherapeutic adjuvants (such as sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine, etc.), it has been found that these remedies are of limited value when actual necrosis of the bone has occurred. The decision to operate early, though superficially it may

TABLE 8.—*Complications of Otitic Infection and Results of Treatment*

	Cases
Perisinal abscess	10
Extradural abscess	7
Meningitis	5
Subperiosteal abscess	6
Thrombosis of the lateral sinus	1
Cholesteatoma	1
von Bezold's abscess	2
Facial palsy	1
Total	33
Results	
Recoveries	28
Deaths (all from meningitis)	5
Total	33
Mortality	15%

appear somewhat radical, must be insisted on even in the absence of any of the usual conclusive positive signs or indications. This point cannot be stressed too strongly. An early operation will save the surgeon many a heartache and will eliminate many of the complications that may develop in an unwitting patient. Usually a thorough simple mastoidectomy is all that is necessary. However, when a perisinal abscess or an epidural abscess is found, a wider exposure of either the lateral sinus or the dura is advisable. Complete eradication of all infected cells is always in order. This may require a rather extensive surgical procedure, since in most cases one is dealing with a well pneumatized mastoid process. The cellular distribution, as has been seen, may be most extensive and may reach throughout the entire squamosa and the zygomatic root into the perilabyrinthine region and to the tip of the petrous pyramid. These areas must be eradicated, since any residual infection in the region will not only prolong the period of convalescence but will also act as a focus from which intracranial complications can develop.

In my series there were 28 cures and 5 deaths (all of meningitis). This gives a mortality rate of 15 per cent, which is comparable to that of Maybaum and Druss (18 per cent) but higher than Vaheri's (10 per cent).

SUMMARY

A study was made of 33 cases of mastoiditis due to Pneumococcus, encountered in the past five years among 190 cases in which positive cultures were reported. A typical clinical picture can be constructed to show the results of a pneumococcic infection. A slow, insidious invasion, characterized by a persistent moderate discharge, which, though purulent, is still rather thin and not under pressure. The drum membrane is somewhat thick, with a satisfactory perforation, and there is very little sagging or narrowing of the canal walls and little or no postauricular edema or tenderness except when a subperiosteal or von Bezold's abscess occurs. Postauricular pain is usually moderate, dull and persistent. Headache, especially hemicrania, is common. The temperature is most often normal or slightly elevated. Roentgen examination reveals extensive pneumatization. This entire picture ordinarily occurs in older patients, and in consequence of all the aforementioned factors shows a marked tendency toward intracranial complications.

CONCLUSIONS

Pneumococcus can produce a typical syndrome, which can account for the deviation of mastoiditis from an assumed normal course.

In a group of 190 mastoids with positive cultures the incidence of pneumococcic infection was 17.3 per cent.

Pneumococcus seems to have a predilection for the extensively pneumatized mastoid bone.

There is an increased frequency of subperiosteal abscess and of von Bezold's abscess.

There is a greater tendency toward intracranial complications, such as perisinal and epidural abscess and meningitis.

When mastoiditis due to Pneumococcus type III is suspected, early operative intervention is definitely indicated.

Late postoperative complications are rare, and the convalescence is usually short and uneventful.

The mortality rate in my series was 15 per cent (all deaths due to meningitis).

All patients should be observed for three to six months after operation, so that any late complication will be recognized at its earliest appearance.

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ABSCESS OF THE MANDIBULAR FOSSA SECONDARY TO OTITIS MEDIA

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Abscesses secondary to otitis media are observed less frequently above and in front of the ear than behind and below the mastoid process. Of abscesses occurring above or in front of the ear, that of the zygomatic fossa is the most common. Abscess of the nasopharynx, which is extremely rare, is the result of extension of infection from the under surface of the petrous pyramid or from the cellular structure of the bone along the eustachian tube. Abscess of the mandibular fossa has been observed only twice at the Mayo Clinic. In neither instance was the lesion situated in the true mandibular fossa, as it was not within the articular space but really caused a subperiosteal elevation of the articular capsule from the surface of the bone. From the observations made it is reasonable to infer that extension of infection from the ear occurred through the glaserian fissure. From the mandibular fossa the infection could easily extend to the pterygomaxillary fossa. That a secondary abscess could occur in the parotid gland by lymphatic extension, as suggested by Hempstead, is shown in our case 2.

ANATOMY

Morris [1] described the mandibular fossa as follows:

> Anterior to the external auditory meatus and separated from it by the tympanic (bone) is a deep cavity, the mandibular fossa, the anterior portion of which, made by the squamous portion of the temporal (bone), is adapted to the articulation of the lower jaw; the posterior part, contributed by the tympanic (bone) lodges an extension of the parotid gland. These two portions are separated by the petrotympanic (Glaserian) fissure, a narrow gap separating the squamous from the tympanic (bones).

The tympanic branch of the internal maxillary artery passes through the fissure, and the chorda tympani passes to the tongue in the canal of Huguier. The structures of the mandibular fossa which are relevant

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From the Section on Otolaryngology and Rhinology of the Mayo Clinic.

1. Morris, H.: Human Anatomy, ed. 7, Philadelphia, P. Blakiston's Son & Co., 1923, pp. 106-140.

to this discussion are best visualized by referring to anatomic specimens or illustrations. From this standpoint the articular capsule of the temporomandibular joint is particularly important. It is often described as consisting of four portions, which are continuous about the articulation. This capsule must be separated from the bone in order to expose the site of the abscess. Entering the real articular surface of the joint should be avoided, for obvious reasons.

Examination of prepared specimens and of roentgenograms discloses a great variation in size of the glaserian fissure. The significance of this opening as a route for the extension of infection had not occurred to us before we made these observations.

REVIEW OF THE LITERATURE

Reports of 5 cases have been found in the literature. In 3 cases the disease was otogenic; in 1 it was of dental origin, and in 1 both aural and dental disease preceded the onset of symptoms. In 1932 Grant [2] made a search of the literature, reviewed the cases of Foster,[3] Strauss [4] and McKenzie [5] and added a case of his own. Greenfield [6] has since added another. In 1908 Foster reported a case of abscess of the pterygomaxillary fossa secondary to dental infection. Infection of the ear was not present. The abscess was drained through the orbit and the sphenomaxillary fissure. The patient did not give a history of dental infection preceding the onset of that which caused the abscess. In 1922 Strauss reported a case in which abscess of the pterygomaxillary fossa complicated acute mastoiditis. Pain, which was centered over the middle third of the zygoma, extended over the entire side of the face and was aggravated by swallowing and by lateral motion of the jaw. Trismus was present. Mastoidectomy was performed, without giving relief; the abscess later ruptured into the mastoid cavity, but counterdrainage, through an incision above the zygoma, was required.

2. Grant, C. C.: Abscess of the Pterygomaxillary Fossa Complicating Otitic Infections: Review of the Literature and Report of a Case, Arch. Otolaryng. **16**:845-850 (Dec.) 1932.

3. Foster, M. L.: Abscess in the Zygomatic Fossa, Ann. Ophth. **17**:439-446 (July) 1908.

4. Strauss, J. F.: Pterygomaxillary Abscess Complicating Acute Mastoiditis, Ann. Otol., Rhin. & Laryng. **36**:477-482 (June) 1922.

5. McKenzie, D.: Otitic Pterygo-Maxillary Abscess Induced by Thrombo-Phlebitis of the Jugular Bulb, Proc. Roy. Soc. Med. (Sect. Laryng.) **16**:53 (Aug.) 1923.

6. Greenfield, S. D.: Pterygomaxillary Abscess Associated with Acute Suppurative Otitis Media Simulating Zygomatic Mastoiditis, Laryngoscope **44**:232-239 (Feb.) 1934.

Strauss considered two possible routes of extension: (1) along the cells of the digastric groove and (2) by rupture through the floor of the petrous bone. Pain was produced on lateral motion of the jaws, because the abscess was under the pterygoid muscles, which produce lateral motion, and by deglutition, because of the insertion of the lateral wall of the pharynx into the base of the petrous bone, which forms part of the roof of the pterygomaxillary fossa. In 1923 McKenzie reported a case of chronic suppurative otitis media with thrombosis of the jugular bulb, which eroded into the pterygomaxillary fossa and formed an abscess. This drained spontaneously through the external auditory canal and into the pharynx. The patient died. In Grant's case the abscess occurred one month after the onset of acute otitis media. There was severe pain in the region of the zygoma, which was not relieved by morphine and was aggravated by swallowing and by moving the jaws. There was a tender mass above the zygoma, and the patient was unable to open his jaws. The abscess was drained through the temporal muscle, and recovery followed. Grant considered the petrotympanic or glaserian fissure to have been the route of extension. In 1934 Greenfield reported a case in which abscess of the pterygomaxillary fossa was associated with resolving acute otitis media and occurred four days after the extraction of a carious upper second molar tooth. Recovery followed external drainage of the abscess. Greenfield expressed the belief that dental infection and the injection of local anesthetic agents for dental extractions were important etiologic factors.

SYMPTOMS AND SIGNS

In the 2 cases of abscess of the mandibular fossa observed at the Mayo Clinic, the symptoms, signs and pathologic changes were definite. The lesion is actually a subperiosteal abscess confined to a small definite space, involvement of which gives the clinical picture. The initial symptom of abscess in the mandibular fossa is severe deep-seated pain in the region of the temporomandibular joint. The pain is increased by motion of the jaw. Fever may be present. Soon severe trismus appears, and swelling of the preauricular structures occurs. If extension to the pterygomaxillary fossa takes place, pain on swallowing may be a symptom. The patient is unable to open the mouth because of trismus and pain. In the early stage the lesion is a subperiosteal abscess without many pathologic changes other than elevation of the periosteum in the small space. In the chronic abscess, fibrous tissue, granulations, burrowing tracts and resorption of bone, producing epidural abscess, may be present. It is possible that this clinical syndrome occurs without involvement of the mastoid cells, by direct extension forward from the middle ear through the glaserian fissure.

DIFFERENTIAL DIAGNOSIS

Abscess of the mandibular fossa must be distinguished from abscess of the temporal fossa (zygomatic). In both, the manifestation of extension, so far as swelling of tissues is concerned, is similar. The pain of the so-called zygomatic abscess is not so severe as the pain of abscess of the mandibular fossa. Trismus from abscess of the zygomatic fossa is a minor complaint compared with that from abscess of the mandibular fossa. Roentgenologic examination usually will give evidence of the presence of mastoid cells beyond the anterior root of the zygoma in cases of abscess of the zygomatic fossa, but it does not necessarily do this in cases of abscess of the mandibular fossa. Inspection of the roentgenogram for the glaserian fissure may be helpful. If the fissure can be seen definitely, it may be inferred that it is patent. It would be possible for infection to extend to both the mandibular and the temporal fossa in cases in which a certain anatomic arrangement of the structure is present.

REPORT OF CASES

CASE 1.—A boy aged 15 years presented himself at the Mayo Clinic in October 1936. He complained of drainage from the ears, which had begun early in the course of severe scarlet fever, three months previously. Myringotomy had been performed on both ears. He had been very ill, with high temperature, prostration and other acute manifestations. Bilateral mastoid operations were performed in the fourth week of the disease. Convalescence was satisfactory, although the mastoid wounds continued to drain and several times small pieces of sequestrated bone appeared in the drainage. In the middle of the second month of illness, severe clonic convulsions, with unconsciousness and injury to the tongue, occurred. Another such convulsion occurred in September.

Examination on entrance revealed that both mastoid wounds were open. Exuberant granulations and small pieces of sequestrated bone were present. The physical and neurologic examinations and a study of the visual fields and the fundi of the eyes gave negative results. Secondary mastoid operations were decided on and performed. On the right side the mastoid cavity was found to be filled with exuberant granulations. No cellular elements were present, but the tegmen tympani was definitely red and diseased. This was removed widely. The dura was seen to be thick, red and coursed by full vessels. It was exposed widely until a more normal type of dura was encountered. No cells were found in the zygomatic region. On the left side, the findings were the same except that the dura was not involved as widely as on the right side. The immediate convalescence was uneventful, and the patient was dismissed from the hospital on the ninth day.

In November, while ambulatory, the patient had an attack of petit mal, which lasted only a few seconds. The second week in November he complained of severe pain on motion of the right mandible. There were fever of 102 F., swelling in front of and above the right ear and definite trismus. The patient was hospitalized, and roentgen therapy was given. This caused a marked increase in the symptoms. On the twelfth day following the onset of the complication an exploratory operation of the right zygomatic fossa was performed. No pus was found. The articular ligament of the mandibular fossa was incised along the lower margin of the

zygoma, and the articular structures were separated from the bone. This permitted the escape of pus from the underlying structures, and pressure on the mass in front of the ear resulted in evacuation of a large quantity of pus. The tract underneath the parotid gland could be visualized, as could the tract under the petrous portion of the temporal bone, in the direction of which the abscess had extended. Appropriate drainage was instituted. After this surgical procedure there was marked general improvement. The trismus lessened, the swelling subsided and on the eleventh day the drainage tubes were removed, since there was but little drainage from the cavity. On the sixteenth day following the exploratory operation, the trismus and pain increased, and the tract was reopened and kept open and draining for a longer period than seemed necessary. From that time improvement was rapid.

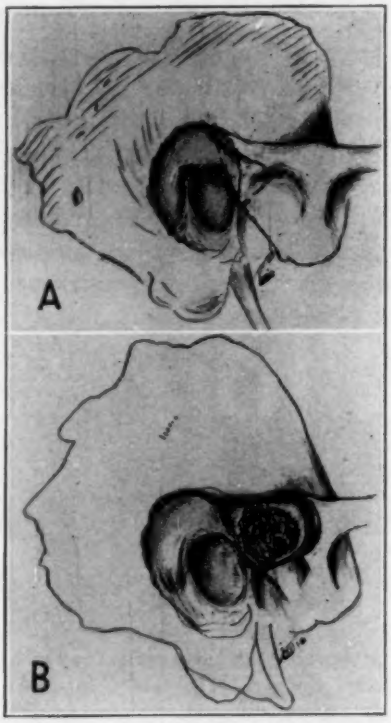
Comment.—We expected to relieve the symptoms by draining a zygomatic abscess, but such an abscess was not found. Extension of the operation to reveal the source of the infection was carried out. With considerable hesitation the articular capsule of the mandibular fossa was incised and separated from the under surface of the zygoma, in order to reach the abscess known to be present. The cavity formed by the abscess at the site of origin was so adequately exposed that it was not deemed necessary to use counterdrainage. The abscess had burrowed under the parotid gland toward the pterygomaxillary fossa.

CASE 2.—A woman aged 32 first presented herself at the Mayo Clinic in August 1935, complaining of earache in the right ear of six weeks' duration. Aural drainage had been present for three weeks. For six weeks she had been unable to open her mouth without severe pain. The past history was irrelevant except for the fact that she had had otitis media in the right ear in childhood. The present condition developed when the patient was on a motor trip, at which time she first noticed difficulty in opening the mouth. This was soon followed by deep-seated pain in front of the ear. Occasionally there were shooting pains into the mastoid region. Tenderness in front of the ear developed quickly, and drainage occurred spontaneously without much relief of the pain.

Examination revealed that the patient was unable to open her mouth more than ½ inch (1.27 cm.) Small tonsillar tags were present in the upper poles of the fossae. The nasopharynx gave no evidence of neoplasm or abscess. The left ear was normal. On the right side there was marked tenderness over the mandibular fossa but no definite swelling. The drum membrane of the right ear was scarred. There was no drainage. A test of the hearing revealed deafness of the type caused by fixation of the stapes. The condition was thought to be arthritis of the mandibular joint. Physical therapy was instituted, without giving much relief. Four days after the first observation, foul pus appeared in the ear, and it was difficult to locate the perforation in the drum through which the pus must have escaped. After this the physical therapy gave marked relief. The patient had to leave our care to carry on her occupation as a school teacher, but she was warned that the trouble was not over.

She was next seen in November 1936. In the interim the symptoms had increased markedly. She had had chiropractic treatment without relief. An exploratory operation on the mastoid process had been performed because drainage from the ear had recurred. The operation had given no relief from the symptoms.

At the time of examination a definite circumscribed swelling was present at the angle of the jaw. This was not fluctuant. There was definite swelling over the root of the zygoma, with marked tenderness on palpation. There was a fistulous tract in the fornix of the right tonsillar fossa. A small amount of pus was expressed on probing, but the probe did not extend into the fossa for any great distance. There was no pharyngeal swelling. Drainage from the middle ear was through a perforation in the anterior portion of the drum membrane. A diagnosis was made of chronic abscess of the mandibular fossa with extension to the pterygomaxillary fossa, and an exploratory operation was advised, but the patient did not act on this advice.



Drawings related to case 2. In *A* the broken line indicates the approach to the abscess under the mandibular fossa. *B* illustrates the epidural abscess encountered.

On Jan. 4, 1937 she returned because she had had a severe exacerbation of all the symptoms two days previously, which had been relieved by copious drainage from the ear. On January 5, through an exposure used for radical mastoidectomy, the mandibular fossa was approached. There was no cellular structure at the zygomatic root. The mandibular fossa was approached by incising the articular ligament along the lower border of the middle root of the zygoma and separating the articular structures from the bone (fig., *A*). Pressure over the mass in front of the ear caused a drop of pus to present at about the position of the glaserian fissure. Further removal of the middle root of the zygoma downward, inward and forward exposed a chronic abscess in the mandibular fossa (fig. *B*). It was

found that the bone forming the upper part of the fossa under the temporal lobe had been resorbed, and the dura was covered with dense granulations. A tract led toward the pterygomaxillary fossa, but none led toward the mass at the angle of the jaw. The dura was exposed in all directions beyond the granulations to normal-appearing dura. The cavity was packed and drained in a suitable manner. It was felt that the mass at the angle of the jaw could be an enlarged gland, and it was not disturbed.

The convalescence was uninterrupted. The symptoms were relieved immediately. After two weeks influenza developed, and a bleb in the left drum membrane caused considerable pain. This cleared up in a day or two, however. The mass at the angle of the mandible had disappeared, so the probabilities are that it had been an enlarged gland which had not suppurated.

Comment.—Whether the otitis media followed suppuration of the mandibular joint or the abscess followed the otitis media in this case is difficult to say. The onset was unlike that of acute otitis media. The abscess was not situated within the joint but was extra-articular. For that reason it is more probable that it originated from extension of infection. The infection appeared to have extended through the glaserian fissure, for it was extra-articular and, further, did not extend below the fissure. The insidious development of the abscess had caused resorption of the bone over the dura, resulting in an epidural abscess. It is not probable that this was a form of petrositis. The duration of the symptoms in this case was unusual. The exploratory operation on the mastoid performed elsewhere had not been extended sufficiently to uncover the lesion. The probable reason was that no zygomatic cells were encountered. As a matter of fact, there was no apparent preformed tract. In accordance with these observations, the abscess in this case, as that in case 1, seems to have been the result of a direct extension of infection of the middle ear through the glaserian fissure to the mandibular fossa. The chronic abscess developed because there had been no drainage.

TREATMENT

The treatment of choice for abscess of the mandibular fossa secondary to otitis media seems to be direct primary surgical exposure of the mandibular fossa, because it is believed that the abscess originates in the mandibular fossa and spreads to the pterygomaxillary fossa. It is obvious that a direct approach at the point of origin of the abscess is preferable to external drainage of a secondary extension. Manifestly, also, it is advisable to drain the abscess before resorption of the bone over the dura takes place. From experience in the 2 cases reported here, it was learned that the approach to the mandibular fossa can be purposeful and direct. The incision employed for a mastoid operation is extended above and around the upper attachment of the ear. Cutting into the temporal muscle is avoided, after this muscle has been exposed,

by making an incision to the bone along the lower aspect of the zygoma to a point just beyond the articular eminence of the mandibular fossa. The temporal muscle and fascia are freed upward sufficiently, and the structures below are separated downward to expose the anterior wall of the bony canal and the curve of the middle root of the zygoma, which forms the fossa on the inner aspect. With a sharp chisel or gouge, a wedge of bone is removed in the direction of the middle ear to the site of the opening of the eustachian tube into the middle ear. The articular capsule is then separated from the mandibular fossa with a suitable periosteal elevator. The field is held open by retractors, and the abscess is thus exposed and dealt with surgically as the conditions found seem to indicate. Suitable drains are inserted, and the upper part of the wound is dressed and kept open until healing takes place. If the abscess has extended to the pterygomaxillary fossa, the question of secondary through and through drainage will need to be settled at the time. If secondary drainage is deemed necessary, an incision involving the parotid gland must be avoided because of the danger of persistent parotid fistula. Means for secondary drainage may need to be provided, depending on the extent of the involvement. The exact position of such drainage would depend on the conditions encountered.

The surgical attack in case 1 was in the nature of an exploration. It happened that the lesion was uncovered at its point of origin, and it seems that such attack is preferable to external drainage, because the lesion can be visualized and dealt with directly. The rationale of direct attack is well demonstrated in case 2, because a chronic abscess with extensive pathologic changes was found. External drainage would not have uncovered the extensive diseased area. The epidural abscess encountered was dealt with as any such abscess encountered in a mastoid operation would be by removal of sufficient bone to uncover the barrier of the abscess or normal dura.

SUMMARY AND CONCLUSIONS

From the experience gained in these cases it is felt that extension of infection from the middle ear to the mandibular fossa takes place through the glaserian fissure. This observation is in agreement with Grant's suggestion. In the 2 cases reported, the symptoms and signs of abscess in the mandibular fossa were identical and definite. A suggested method is outlined for making a direct surgical approach to the abscess in the mandibular fossa rather than relying on drainage of secondary extensions of the abscess.

LEIOMYOSARCOMA OF THE LARYNX

REPORT OF A CASE

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A malignant growth of a sarcomatous nature involving the larynx is of rare occurrence, and particularly rare is leiomyosarcoma of the larynx. Only 1 case of laryngeal leiomyosarcoma has been reported in the literature.[1] Figi,[2] in reviewing 717 cases of malignant disease of the larynx encountered since 1910 at the Mayo Clinic, noted only 4 cases of sarcoma in this group—a percentage of 0.79. Kahler[3] had seen only 2 cases of sarcoma in his own practice during a period of twenty-seven years and had found only 183 cases reported in all the literature up to 1928. Sendziak[4] reported that there is 1 carcinoma of the larynx to every 30 carcinomas. Semon[5] saw 3 sarcomas among 103 malignant growths of the larynx. The Jacksons[1] in their new book reported only 5 cases of sarcoma. Imperatori[6] encountered only 3 cases in all the years of his practice.

Sarcomas are malignant tumors derived from various connective or supporting tissues of the body. Their classification into varieties depends on the amount of connective tissue stroma, on the relationship of tumor cells to blood vessels, on the type of interstitial tissue (fibrous, bony or cartilaginous), sometimes on the shape of the cells and, finally, on certain chemical properties of the cells which lead them to produce pigment (melanosarcoma, chloroma). Sarcoma is more frequent in the skin, subcutaneous and subserous connective tissue, fascia, marrow, periosteum of bone and choroid coat of the eye. Common sites are the uterus and the ovary. As indicated in the preceding paragraph, sarcoma constitutes less than 1 per cent of all malignant laryngeal neoplasms. The different histologic forms reported as appearing in the larynx are fibrosarcoma, myxosarcoma, chondrosarcoma, angio-

1. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939.

2. Figi, F. A.: Sarcoma of the Larynx, Arch. Otolaryng. **18**:21 (July) 1933.

3. Kahler, O., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 5, p. 483.

4. Sendziak, cited by Kahler.[3]

5. Semon, cited by Kahler.[3]

6. Imperatori, C. J.: Personal communication to the author.

sarcoma, melanosarcoma, lymphosarcoma, rhabdomyosarcoma, and, in 1 case, leiomyosarcoma. There are also mixed forms, as well as carcinosarcoma.

Sarcoma is usually a primary tumor, but metastatic deposits occurring in the larynx have been reported. Extensions of sarcomatous growths of the tongue, pharynx, thyroid and trachea have also been reported. Sarcoma occurs in any part of the larynx, but more frequently on the vocal cord. It is usually a circumscribed lobulated smooth growth and occasionally is pedunculated. The color is variable, more prone to be yellow than red, depending on the contained blood vessels. It may be light gray, light red, blue-red or wine yellow. The consistency depends on the type of histologic structure, and the tumor varies in size from that of a lentil to that of a chicken egg. Sarcoma has little tendency to infiltrate and metastasize. Ulcerations are infrequent, in contradistinction to the frequency seen in carcinoma. Sarcoma usually grows more slowly than carcinoma.

Leiomyosarcoma is a rare tumor in any part of the body. It has been reported in the female genitourinary tract, in the gastrointestinal tract and in the skin. It is most frequently found in the uterus and is said to occur in 1 per cent of myomatous uteri.

The treatment of sarcoma of the larynx depends on the location, size, type and activity of the growth. The tumor may be removed through the laryngoscope, by mirror laryngoscopy, by thyrotomy, by laryngectomy, by pharyngotomy, by electrocoagulation or by roentgen and radium therapy.

It is important that, while the case to be reported here is being reported as an instance of leiomyosarcoma of the larynx, there was a divergence of opinion regarding this diagnosis. Although the consensus was in favor of such interpretation, the diagnosis of a neurogenic tumor—neurogenic fibroma or neurosarcoma—was also made.

With regard to the possibility of a neurogenic origin of this tumor, I find that a growth of such origin is almost as rare as leiomyosarcoma. Figi [7] offered the following description of neurogenic tumors:

> Grossly the tumors are rounded and often nodular. They are encapsulated and may be hard or cystic. . . . They are always attached to a nerve or a nerve root. . . . The attachment to a nerve may not be demonstrable owing to inadequate exposure and to the vascularity of the adjacent tissues. The color is usually distinctly brownish or yellowish.
>
> Such tumors arise from the sheaths of nerves or from nerve roots. They are most commonly situated centrally, that is, on nerve roots, in contradistinction to the tumors of neurofibromatosis which are, as a rule, found peripherally.

7. Figi, F. A.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryng. **17**:386 (March) 1933.

Penfield [8] has given the name "perineural fibroblastoma" to solitary neurofibroma and has directed attention to the bewildering number of names applied to this tumor as evidence of confusion in the minds of the many clinicians who have studied the neoplasm. Among the terms used may be mentioned "solitary neurofibroma," "neurinoma," "fibroma of nerves," " gliofibrosarcoma," "peripheral glioma," "schwannoma," "neurogenic fibroma" and "tumor of the acoustic nerve." New and Erich,[9] reviewing 722 cases of benign tumor of the larynx encountered at the Mayo Clinic during the past thirty years, reported but 1 case of neurofibroma of the larynx. The patient was a 49 year old woman who had intermittent hoarseness for twelve years. The tumor was attached to the free border of the middle third of the right vocal cord. Jackson and Coates [10] reported a neurofibroma in a woman 23 years of age. They declared neurofibroma to be a very rare tumor. The location in the larynx was not stated, but it is noted that there had been no associated visceral or dermal lesions of the same character as those seen in neurofibromatosis. Figi [7] reported a solitary neurofibroma of the pharynx. He stated that tumors originating from the nervous system or containing nerve elements and appearing primarily within the oral cavity or pharynx are rarely encountered but may occur in association with generalized neurofibromatosis (von Recklinghausen's disease). He reviewed the literature and failed to find a report of another solitary neurofibroma primary in the pharynx. Vail [11] reported a case of schwannoma of the larynx, a large round pinkish red tumor attached to the right aryepiglottic fold, which was removed by Lynch-Killian suspension laryngoscope. He included in his paper a report of 2 similar cases appearing in the literature, in which a schwannoma was attached to the aryepiglottic fold.

REPORT OF A CASE

Mrs. P. W., a housewife aged 28, presented herself on March 7, 1939 with the following complaints: choking sensation, moderate difficulty in swallowing, a harsh voice and occasional pain in the right ear. These symptoms were first manifested about three years previous to her initial visit to me and had become progressively worse during this period. The personal history was essentially negative. The patient is a member of a strongly allergic family. She is married and has two children, one 5 years of age and the other 8 months. Examination

8. Penfield, W.: The Encapsulated Tumors of the Nervous System, Surg., Gynec. & Obst. **45**:178 (Aug.) 1927.

9. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty-Two Cases, Arch. Otolaryng. **28**:841 (Dec.) 1938.

10. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

11. Vail, H. H.: Schwannoma of Larynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:418, 1933.

of her throat showed that the tonsils had been successfully removed. The mucous membrane of the pharynx was moderately congested, and there was a small amount of mucoid secretion in the postnasal space. Examination of the nose showed the septum to be deviated to the left and the turbinates moderately hypertrophied. The ears were normal. Mirror examination of the larynx revealed a large circumscribed smooth globular tumor (fig. 1 *A*), somewhat fissured, which hung like a curtain in front of the larynx and apparently had its origin on the laryngeal surface of the epiglottis. It was light red and was about the size of a hazelnut. Only the posterior portion of both vocal cords could be seen. The heart and lungs were normal, and the blood pressure was 115 systolic and 70 diastolic. The urine showed clumps of pus cells. The Wassermann reaction was negative and the blood count did not deviate from the normal.

The appearance of this tumor, together with the history of three years' duration, suggested that the growth was probably benign. Dr. Charles J. Imperatori, who saw this patient in consultation, concurred in this opinion. We decided that the mass should be removed through a Jackson laryngoscope with the patient under general anesthesia. We also gave consideration to other methods of approach,

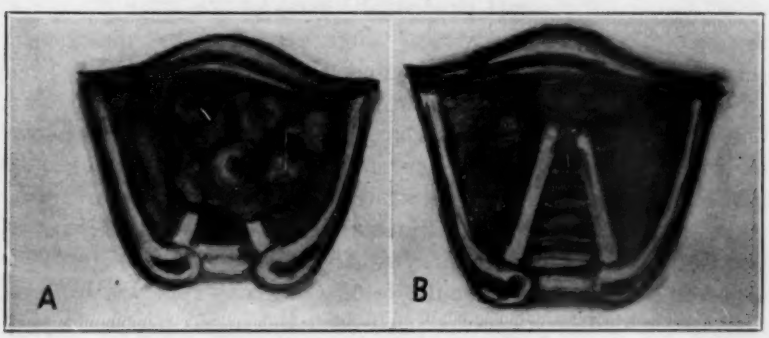

Fig. 1.—*A*, the tumor as seen by mirror examination. *B*, the larynx after operation, showing a small portion of the tumor which escaped removal.

viz., suspension laryngoscopy, thyrotomy and indirect laryngoscopy, but we felt that direct laryngoscopic removal would be best.

On March 15 this procedure was carried out at the New York Polyclinic Medical School and Hospital with the assistance of Dr. Imperatori. Because of the location of the mass on the laryngeal surface of the epiglottis, the removal was a difficult technical procedure. Only a small rim of the epiglottis was free of the tumor. This made the lifting of the epiglottis with the beak of the laryngoscope trying. The laryngoscope either slipped off the epiglottis or encroached on the tumor to hide it from view. However, by careful manipulation the tumor was removed with a biting forceps. Examination of the patient a few days later revealed the presence of a small portion of tumor which had escaped removal (fig. 1 *B*).

Dr. A. S. Price, pathologist to the New York Polyclinic Medical School and Hospital, reported the examination of tissue as follows: "Microscopic examination reveals a portion of mucous membrane from the region of the larynx, beneath which is a cellular tumor showing two types of elements. One is rather cellular and follows closely the characteristics of a leiomyoma. These cells are fairly well

differentiated, and there is a slight excess of collagenous fibrils, which may mean that the tumor is of neurogenic origin or is a very cellular fibroma. There are no positive signs of malignant disease. The second element is more of connective tissue type, with a tendency toward mucinous degeneration. The cells and nuclei are very small, with an abundance of interstitial tissue. Diagnosis: leiomyoma (?) of the larynx." Dr. Price suggested that Dr. James Ewing see the slide which he had examined, and Dr. Ewing reported as follows: "Dr. F. W. Stewart and I agree with Dr. Price that this is a leiomyosarcoma. The only other possible

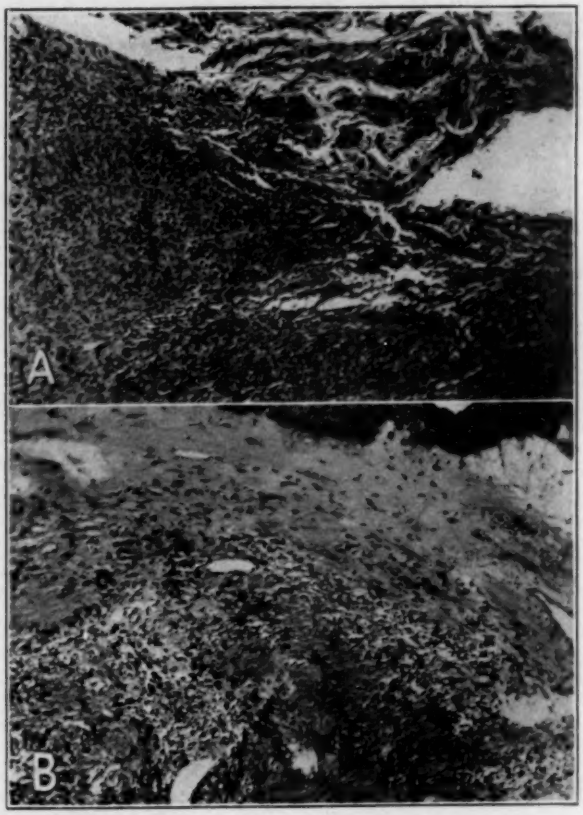

Fig. 2.—*A*, general view of the tumor tissue. *B*, low power view, showing surface epithelium. The tumor is in the lower half of the field. Note the tendency of the nuclei to group into palisades.

diagnosis is neurosarcoma, which we exclude. The process is rather cellular, and while it is somewhat circumscribed, it is not encapsulated, and I fear it would be difficult to remove surgically. I do not know that any one has used radiation on such a tumor in the larynx, and I cannot predict the reaction to radiation. I am inclined to think that the tumor may be rather radiosensitive. I suggest roentgen rays transmitted through a brass cylinder just covering the lesion and in doses short of those required for cancer but sufficient to cause sharp blistering of the skin. If this is not carried too far, the treatment would show whether the tumor is sensitive and would not greatly influence subsequent surgical procedure.

I fear that you will see a recurrence after any but a radical operation and will then be compelled to rely on radiation under much less favorable circumstances. While this tumor is cellular, I doubt if it will produce metastases."

In accordance with the suggestion made by Dr. Ewing, the patient was referred to the radiotherapy department of the Montefiore Hospital for Chronic Diseases for high voltage roentgen therapy to the remaining portion of the tumor, which was approximately the size of a large pea. The course of roentgen therapy was begun on April 10 and was completed on May 15. The patient received 3,350

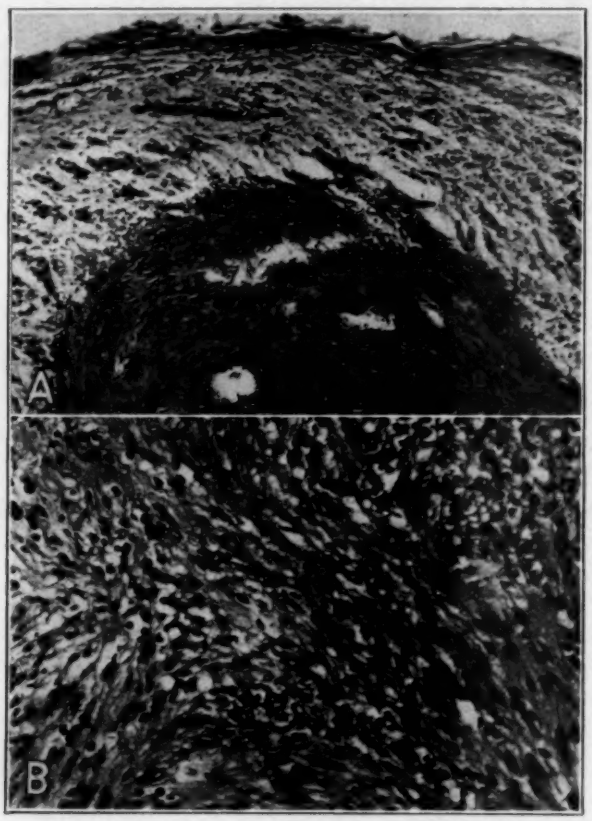

Fig. 3.—*A*, low power view of the edge of a tumor nodule. Note the strands radiating into surrounding connective tissue. *B*, medium power view, showing cellular detail. Note mitotic figure, lower center.

roentgens (r) with back scatter to each side of the larynx. The other factors were: current, 200 kilovolts 25 milliamperes; a Thoreus filter equivalent to 2 mm. of copper plus 1 mm. of aluminum; skin-target distance, 80 cm. The roentgen therapy did not influence the growth in any way. It remained stationary in size.

On October 19 the remaining portion of the tumor on the laryngeal surface of the epiglottis was removed with a biting forceps by the indirect method. The report of examination of this issue is as follows: "The specimen consists of a portion of tumor nodule 1 cm. in diameter, said to have been taken from the larynx. Microscopic examination shows a portion of tumor made up of mucinous

tissue and smooth and involuntary muscle. The nuclei are fairly large, and cytoplasmic borders are difficult to outline. The tumor is slightly more cellular now than on previous examination. It is my impression that any therapy that has been given has been of no particular benefit, and, as frequently occurs with this type of tumor, the growth more nearly approaches a malignant state now than it did in the original specimen. Diagnosis: leiomyosarcoma of the larynx." (Figs. 2 and 3.)

In preparing this case report for publication, I prevailed on Dr. Joseph Ehrlich, pathologist to Lebanon Hospital, to make photomicrographs for me. On reviewing the slides, he suggested that the tumor might be of neurogenic origin. This opinion was upheld by Dr. Sadao Otani, of the Mount Sinai Hospital. The slides were then returned to Dr. Price for examination, and he too favored the diagnosis of neurogenic tumor.

I then requested Dr. Ewing to review these slides together with sections of tissue specially prepared with nerve stains. The following is his final report:

"I have reviewed the old sections and all the new sections from the larynx in the case of P. W., and they have been reviewed by Drs. Stewart and Foote, of Memorial Hospital, and by Dr. Chandler Foot, of the New York Hospital, who is an expert in the use of nerve stains. There is considerable new material in the later sections, not present in the first section submitted. The result of all these studies shows that the prevailing opinion in this laboratory is toward the diagnosis of leiomyosarcoma. The most suggestive feature pointing to neurosarcoma is the presence of a few islands of palisade cells, such as occur in neuroma. We feel that somewhat similar conditions are seen in muscle tumors, and they are not very distinct in this tumor. Moreover, there are numerous areas in the tumor where the cells seem to have the characters of elongated smooth muscle. Some of these are shown in the photographs. The mucous characters surrounding some lobules occur in both nerve and muscle tumors. I have to confess that I am still in doubt as to the exact nature of this process, being considerably impressed by the presence of palisade cells, which definitely suggest neurofibroma. Yet I am not willing to take a position contrary to that reached by numerous colleagues. Dr. Price and I rather favor the diagnosis of neurogenic fibroma, and the other pathologists feel rather sure that the tumor is myogenic."

Examination of the patient's larynx on Nov. 12, 1940 failed to reveal any evidence of recurrence. She now has a good speaking voice and does not complain of any local laryngeal discomfort.

SUMMARY

The case reported here presents the following interesting features:

1. Rarity of sarcoma of the larynx. Approximately 1 per cent of all malignant growths of the larynx are sarcomas, and leiomyosarcoma is exceedingly rare.

2. Pathologic controversy. This tumor presented characteristics suggestive of both myogenic and neurogenic origin, but the consensus among the pathologists who examined the tissue favored the diagnosis of leiomyosarcoma.

3. Approach to the tumor. This growth, situated on the cushion of the epiglottis, presented the problem of satisfactory approach and removal. Consideration was given to suspension laryngoscopy, thyrotomy and amputation of the epiglottis with the attached tumor, but the simple method of direct laryngoscopic removal with a biting forceps was utilized, with almost complete success. A small portion of the tumor escaped removal, but it was subsequently successfully removed by mirror laryngoscopy. It is interesting to note that Dr. Ewing suggested that only the most radical surgical procedure ought to be utilized, lest there be a recurrence. However, the least radical was carried out, and there is no evidence of recurrence after eighteen months.

4. Effect of radiation. The small portion of tumor which remained after the initial surgical procedure through the laryngoscope was subjected to radiation, but the treatment did not influence the size of this remaining tissue.

78 Irving Place.

REVERSAL OF HALISTERESIS IN THE MAMMALIAN EAR

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AND

MARY IRVINE, A.B.

LOS ANGELES

In otosclerosis one deals with an osseous dystrophy of the aural capsule and in many cases with a neuropathy of the eighth nerve. Until recently the neural degenerative changes (fig. 1 *A*) were not considered an essential part of the pathologic picture of progressive hereditary deafness but were regarded rather as a complication due to other factors.

Surrounding the focus of otosclerotic bone is usually an area of decalcification, or halisteresis (fig. 1 *B*). This area of "preparedness" finally becomes, through complete bone resorption (fig. 2 *A* and *B*), a cavity filled with osteogenetic marrow, derived from the focus of dystrophy. In this marrow, new abnormal bone forms, thus increasing the size of the original focus.[1]

A series of patients with classic otosclerosis have been treated with dietary regulation and administration of dicalcium phosphate ($CaHPO_4$), vitamins, insulin, extract of liver and other preparations and observed over a period of four years. These patients have shown no further loss of hearing.

Some of the patients treated have an improvement in hearing. The improvement, we presume, has resulted from administration of thiamine hydrochloride, nicotinic acid, insulin and extract of liver. With the improved patients we are not concerned here.

A possible explanation of the absence of further deterioration in hearing may be found, we believe, in the reversal of halisteresis in the aural capsule—a rebuilding of osseous tissue around the focus of otosclerosis. Such a reossification would tend to inhibit further increase in the size of the dystrophic area (a retaining wall effect).

In order to prove the possibility of reversing halisteresis in the aural capsule, the research reported here was undertaken.

Presented before the Research Study Club, Jan. 26, 1939.

From the Department of Otolaryngology, University of Southern California.

The research reported here was made possible through a grant from the Research Study Club of Los Angeles. The animal experimentation was done in the laboratories of the Children's Hospital.

1. Guggenheim, L. K.: Otosclerosis, St. Louis, The Author, 1935, p. 135.

Halisteresis was produced in Wistar stock white rats with parathyroid extract,[1a] ammonium chloride and viosterol.[1b] A low and a high calcium experiment were included.

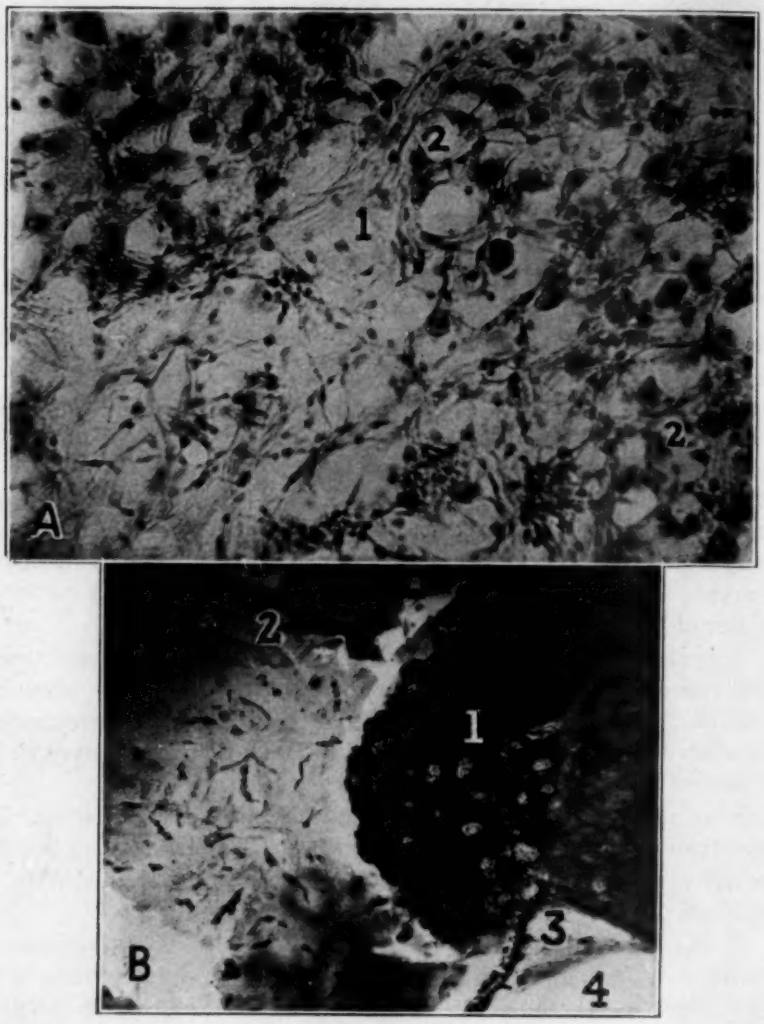

Fig. 1.—*A*, section through the spiral ganglion of a patient with otosclerosis. *1* indicates fibrosis following the disappearance of ganglion cells. *2* indicates vacuoles. *B*, otosclerotic focus at a site of predilection. *1* indicates dystrophic bone; *2*, the capsule; *3*, the fissula ante fenestram, and *4*, the cisterna. (This picture is published by permission of E. W. Hagens.) The use of a magnifying glass will greatly enhance the value of the photomicrographs.

1a. The parathyroid extract used in these studies was a preparation of solution of parathyroid U. S. P. (parathyroid hormone-Squibb).

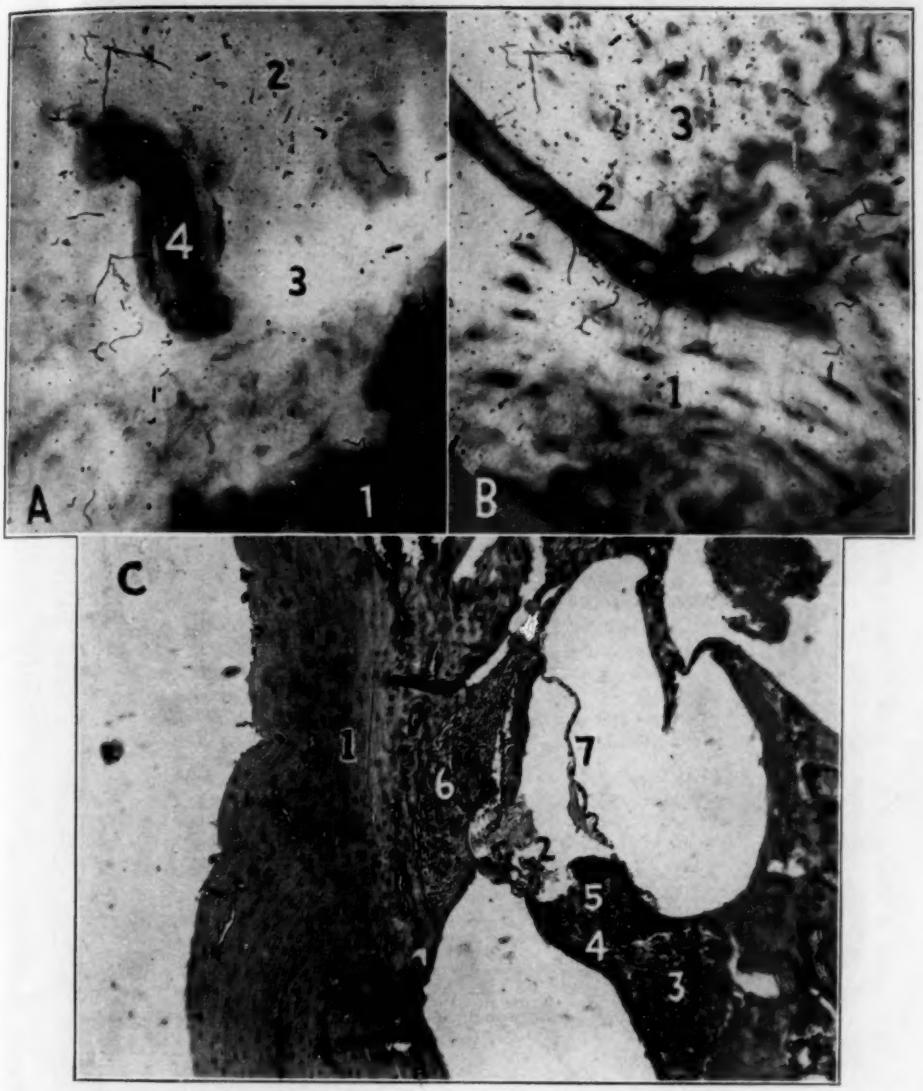

Fig. 2.—*A* and *B*, blue mantles in otosclerosis (picture shown by permission of E. W. Hagens). In *A*, *1* indicates otosclerotic bone; *2*, marked halisteresis of the capsule; *3*, a resorption space, and *4*, the blue mantle. In *B*, *1* indicates halisteresis of the capsule; *2*, the blue mantle, and *3*, the resorption area. *C*, low power photomicrograph of a section through the cochlea of a control rat on the basic diet. *1* indicates the otic capsule, which is normal as to matrix, osteocytes and vessels. *2* indicates the organ of Corti; *3*, the spiral ganglion; *4*, Rosenthal's canal; *5*, the limbus laminae spiralis; *6*, the ligamentum spirale cochleae, and *7*, the membrana vestibularis Reissneri. The membranous cochlea is normal. Since the research concerned itself solely with bone, no attempt was made to get good sections of the membranous labyrinth.

1b. The viosterol used in this work was a preparation of viosterol in oil N. N. R., containing 10,000 units (U. S. P.) of vitamin D in each gram.

TABLE 1.—*Weights (Gm.) of Male Rats 1 to 12 (Experiment in the Production of Halisteresis)*

Date	Rat 1 (45-83)*	Rat 2 (60-118)*	Rat 3 (60-118)*	Rat 4 (60-118)*	Rat 5 (60-118)*	Rat 6 (60-118)*	Rat 7 (45-83)*	Rat 8 (45-83)*	Rat 9 (45-83)*	Rat 10 (45-103)*	Rat 11 (45-83)*	Rat 12 (45-83)*
11/10/37	121	167	191	181	182	142	102	100	110	105	100	115
11/12/37	120	180	200	192	190	150	112	110	118	110	110	120
11/13/37	138	185	207	200	200	165	117	115	122	90	112	127
11/15/37	140	190	210	208	210	175	125	120	130	95	120	120
11/17/37	142	190	213	210	210	178	126	119	133	97	120	120
11/19/37	160	201	214	214	218	200	135	128	140	105	136	120
11/20/37	165	202	218	216	222	201	151	143	143	109		
11/22/37	170	200	220	219	227	207	152	148	146	110	140	121
11/23/37	.....	.....	Taken off basic diet and put on low calcium diet									
11/24/37	180	203	221	218	228	205	154	149	145	111	140	122
11/26/37	.....	.....	.....	.....	Taken off basic diet and put on high calcium diet							
11/27/37	190	208	222	217	226	216	154	147	149	124	144	133
11/29/37	205	209	238	235	245	225	Parathyroid extract (0.5 cc.) given daily from 11/29/37 on		157 Ammonium chloride (250 mg. per day) added to basic diet	132	157 12/3/37: 34,000 units of vitamin D (viosterol) added to basic diet	152
12/ 1/37	215	210	241	231	235	215	157	150	174	145		
12/ 3/37	217	213	246	241	236	215	158	152	177	144	170	165
12/ 5/37	...	...	...	...	.....	.....	.....	.....	.....	.....	169	169
12/ 6/37	221	213	256	251	251	230	175	160	180	158	169	182
12/ 8/37	227	217	245	244	244	215	180	164	179	160	170 On 12/8/37 and 12/9/37, the viosterol was placed directly in the rats' mouths, after which it was again mixed with food	181
12/10/37	229	220	251	250	250	220	185	165	180	165	175	180
12/13/37	230	224	250	250	252	223	186	167	180	166	174	178
12/15/37	240	225	270	265	230	220	187	175	182	167	177	180
12/16/37	.....	.....	.....	.....	250	230						
12/17/37	245	230	280	270	255	235	190†	175†	176†	170	175	179
12/18/37	250†	...	...	...	.....	.....	.....	.....	.....	.....	175†	161†
12/20/37	...	234	284	275	261	239	.....	.....	...	174	.....	.....
12/22/37	...	243	280	278	260	240	.....	.....	...	175	.....	.....
12/24/37	...	245	282	277	262	238	.....	.....	...	174	.....	.....
12/27/37	...	244	281	277	260	239	.....	.....	...	173	.....	.....
12/29/37	...	246	282	279	261	238	.....	.....	...	174	.....	.....
12/31/37	...	251	281	280	260	240	.....	.....	...	178	.....	.....
1/ 4/38	...	260	290	290	260	240	.....	.....	...	200	.....	.....
1/ 7/38	...	261†	286†	294†	266†	250†			...	208† (dead)	.....	.....
	210‡ (250 — 40)	216‡ (261 — 45)	234‡ (286 — 52)	243‡ (294 — 51)	217‡ (266 — 49)	208‡ (256 — 48)	145‡ (190 — 45)	147‡ (175 — 28)	136‡ (176 — 40)	164‡ (208 — 44)	145‡ (175 — 30)	133‡ (164 — 31)

* The first figure indicates the age of the rat in days at the beginning of the experiment, and the second, the age when the rat was killed.
† This figure represents the weight when the rat was killed.
‡ This figure represents the weight of the dead rat minus viscera.

The basic diet given all rats throughout the experiments was as follows: sucrose, 50 parts; casein, 25 parts (contained 0.01 per cent calcium), and hydrogenated cottonseed oil, 25 parts. To each hundred grams of this was added the following salt mixture: precipitated calcium phosphate, 1.5 Gm.; sodium chloride, 0.73 Gm.; potassium chloride, 1.25 Gm.; ferric citrate, 0.13 Gm.; sodium iodide, 0.03 Gm., and magnesium sulfate ($MgSo_4.7H_2O$), 1 Gm.

For the low calcium diet, calcium phosphate was omitted and 3.5 Gm. of sodium phosphate ($Na_2HPO_4.12H_2O$) was added.

For the high calcium diet there was added to the basic diet 2 Gm. of calcium carbonate per hundred grams.

To all diets were added 10 per cent brewers' yeast and 1 per cent cod liver oil.

The weights of the rats are shown in table 1.

The leg bones, femur plus tibia, and skulls of the 12 rats were sent to the Department of Biochemistry, University of California, Berkeley, for analysis. This work was done by Dr. Elma V. Tufts.

TABLE 2.—*Recapitulation of Data in Table 1*

Rat	Diet	Time and Gain in Weight, Gm.	Average Daily Gain, Gm.
1	Basic	38 days, 129	3.3
2	Basic	58 days, 94	1.6
3	Low calcium	58 days, 95	1.6
4	Low calcium	58 days, 109	1.8
5	High calcium	58 days, 54	0.93
6	High calcium	58 days, 114	1.9
7	Parathyroid extract	48 days, 98	2.0
8	Parathyroid extract	48 days, 75	1.5
9	Ammonium chloride	37 days, 66	1.7
10	Ammonium chloride	37 days, 103	1.7
11	Viosterol	38 days, 75	1.9
12	Viosterol	38 days, 49	1.2

The leg bones and the skulls were examined as to weight, ash weight, ash per cent, total calcium, per cent calcium in ash, per cent calcium in bone, total phosphorus, per cent phosphorus in ash and per cent phosphorus in bone.

Table 3 gives the results.

PRIMARY EVALUATION OF ANALYSIS

Weights.—1. Leg Bones (Tibia and Femur): The low calcium diet reduced the weight of a leg from 0.5160 Gm., the minimum on the basic diet, to the minimum 0.449 Gm.—a loss of 0.067 Gm. The high calcium diet produced a gain in weight from 0.516 Gm., the minimum on the basic diet, to 0.609 Gm.—a gain of 0.093 Gm. From the maximum basic diet weight (0.564 Gm.) to the maximum obtained with the high calcium diet there is an increase of 0.132 Gm. The parathyroid minimum compared with the basic diet minimum shows a loss of 0.125 Gm. The ammonium chloride minimum (0.409 Gm.) compared with the basic

diet minimum (0.516 Gm.) shows a loss of 0.107 Gm. The viosterol minimum (0.350 Gm.) compared with the basic diet minimum (0.516) shows a loss of 0.166 Gm.

2. Skulls: A comparison of the miniumum weight of the skulls of rats on the basic diet (0.5065 Gm.) with the minimum weight of the skulls of those on the low calcium diet (0.573) shows an unexpected gain in the latter (0.0665 Gm.).

A comparison of the basic diet minimum (0.5065) with the high calcium minimum (0.705) shows the expected gain in the latter of 0.1985 Gm.

TABLE 3.—*Analysis of Leg Bones (Tibia plus Femur) and of Skulls of Rats Used in Experimental Production of Halisteresis*

			Ash		Calcium			Phosphorus		
Diet	Part Analyzed	Bone Weight (Gm.)	Weight (Gm.)	% of Bone Weight	Weight (Mg.)	% in Ash	% in Bone	Weight (Mg.)	% in Ash	% in Bone
Basic	Leg 1	0.5160	0.309	59.3	94.0	32.1	19.2	46.4	14.9	9.0
Basic	Leg 2	0.564	0.371	71.2	123.5	33.3	21.9	56.8	15.4	10.1
Low calcium	Leg 3	0.535	0.320	63.7	81.0	25.3	15.12	52.3	16.3	9.8
Low calcium	Leg 4	0.449	0.315	72.8	98.0	31.1	21.8	52.6	16.7	11.7
High calcium	Leg 5	0.696	0.431	65.6	134.5	31.3	19.3	72.4	16.7	10.4
High calcium	Leg 6	0.600	0.416	70.7	129.0	31.05	21.2	69.4	16.7	11.4
Parathyroid extract	Leg 7	0.391	0.250	66.7	93.0	37.2	23.8	43.9	17.5	11.2
Parathyroid extract	Leg 8	0.5172	0.304	58.4	95.0	31.2	18.38	49.5	16.3	9.6
Ammonium chloride	Leg 9	0.430	0.284	68.8	94.0	33.1	21.9	47.7	16.8	11.1
Ammonium chloride	Leg 10	0.409	0.287	72.4	91.0	31.7	22.2	48.1	16.8	11.8
Viosterol	Leg 11	0.351	0.264	76.8	85.5	32.3	24.3	44.9	17.0	12.8
Viosterol	Leg 12	0.350	0.240	70.4	79.5	33.1	22.7	41.2	17.2	11.8
Basic	Skull 1	0.5065	0.403	79.6	106.8	26.5	21.1	63.7	15.7	12.5
Basic	Skull 2	0.788	0.470	65.6	137.5	29.3	17.4	69.0	14.0	8.8
Low calcium	Skull 3	0.673	0.385	59.8	103.5	26.9	15.4	58.8	14.7	8.7
Low calcium	Skull 4	0.573	0.380	69.2	98.5	25.9	17.2	52.6	13.8	9.0
High calcium	Skull 5	0.741	0.497	70.4	136.5	27.5	18.4	71.5	14.4	9.6
High calcium	Skull 6	0.705	0.492	71.6	136.0	27.7	19.3	75.8	15.4	10.7
Parathyroid extract	Skull 7	0.389	0.253	65.0	196.5	38.1	24.8	39.9	17.1	10.3
Parathyroid extract	Skull 8	0.5957	0.330	60.4	137.0	41.5	25.1	56.5	17.1	10.2
Ammonium chloride	Skull 9	0.382	0.270	72.6	84.8	30.3	22.2	40.9	15.1	10.7
Ammonium chloride	Skull 10	0.570	0.361	64.8	117.2	32.3	20.7	54.9	15.2	9.6
Viosterol	Skull 11	0.329	0.234	71.0	76.0	33.1	23.1	37.5	16.3	11.4
Viosterol	Skull 12	0.380	0.234	70.7	79.5	30.55	20.9	41.0	15.8	10.8

The parathyroid minimum (0.389) shows a loss over the basic diet minimum (0.5065) of 0.1175 Gm.

The ammonium chloride minimum (0.382) shows a loss over the basic diet minimum (0.5065) of 0.1245 Gm.

The viosterol minimum (0.329) shows a loss over the basic diet minimum (0.5065) of 0.1775 Gm.

The greatest loss, then, was from viosterol.

Calcium and Phosphorus Contents (table 4).—1. Calcium: The amounts of calcium recovered from the leg bones are quite in accord with what one would expect. The enormous dose of vitamin D with

insufficient calcium to balance caused the most marked mobilization and loss of calcium from these bones.

The skulls of the rats which were given the viosterol diet showed the lowest calcium content. Of the three decalcifying agents, parathyroid produced the least loss of calcium.

2. Phosphorus: The lowest phosphorus content observed in the leg bones, like the lowest calcium content, was associated with the vitamin D diet. Interesting is the highest phosphorus recovery, 70.9 mg., which occurred with the high calcium diet.

Unlike the leg bones, the skulls of the rats which were given the low calcium diet showed a considerable drop in phosphorus content. Again, the lowest phosphorus content was found in the skulls of the animals which had been fed viosterol. The high calcium diet brought the phosphorus deposit in the skulls up materially. As in the legs, the three decalcifying agents produced the greatest drop in phosphorus content.

TABLE 4.—*Average Amounts of Calcium and Phosphorus (Mg.) Recovered from Leg Bones and Skulls*

Diet	Calcium		Phosphorus	
	Leg Bones	Skulls	Leg Bones	Skulls
Basic	108.75	122.15	51.6	66.35
Low calcium	89.50	101.00	52.45	55.70
High calcium	131.75	136.25	70.9	73.65
Parathyroid extract	94.00	116.75	46.7	48.20
Ammonium chloride	92.50	101.00	47.9	47.90
Viosterol	82.50	77.75	43.05	39.25

ANALYSIS OF DATA IN TABLE 5

In the evaluation of the loss or gain in calcium and phosphorus in the various bones examined, it must be kept in mind that the animals treated with ammonium chloride and other agents did not gain in weight as did the controls; hence, the amounts of calcium and phosphorus obtained in the experiment must be computed on the basis of the comparative size and age of the animals.

Low Calcium Experiment.—The body weights of the animals which were given the low calcium diet were greater than those of the controls (table 5, column 3), but the bone weights were smaller (column 5). The absolute amounts of calcium in the ash (column 10) were less, and the absolute amounts of phosphorus (column 14) were slightly reduced.

The skulls were smaller than those of the controls (column 5) and the ash weights were reduced (column 7). The absolute amounts of calcium (column 10) and of phosphorus (column 14) in the ash were reduced.

TABLE 5.—*Further Analysis of Leg Bones (Tibia plus Femur) and Skulls of Rats Used in Experimental Production of Halisteresis with Account Being Taken of Age and Weight of Rats*

Diet	Age of Rat at Death, Days	Body Weight, Gm.	Part Analyzed	Weight of Bone, Gm.	% of Body Weight	Ash Weight			Calcium				Phosphorus			
						Gm.	% of Body Weight	% of Bone Weight	Mg. in Ash	mM	% in Ash	% in Bone	Mg. in Ash	mM	% in Ash	% in Bone
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)
Basic	118	261	Leg 2	0.564	0.216	0.371	0.142	71.2	123.5	3.08	33.3	21.9	56.8	1.84	15.4	10.1
Low calcium	118	286	Leg 3	0.535(—)	0.187	0.320	0.112(—)	63.7	81.0	2.02	25.3	15.12	52.3	1.69	16.3	9.8
Low calcium	118	294	Leg 4	0.449(—)	0.153	0.315	0.107(—)	72.8	98.0	2.45	31.1	21.8	52.6	1.70	16.7	11.7
High calcium	118	266	Leg 5	0.696	0.262	0.431	0.162	65.6	134.5	3.36	31.3	19.3	72.4	2.34	16.7	10.4
High calcium	118	256	Leg 6	0.609	0.238	0.416	0.167	70.7	129.0	3.22	31.05	21.2	69.4	2.24	16.7	11.4
Ammonium chloride	108	208	Leg 10	0.409	0.197	0.287	0.138	72.4	91.0	2.28	31.7	22.2	48.1	1.55	16.8	11.8
Basic	83	250	Leg 1	0.516	0.206	0.309	0.120	59.3	94.0	2.35	32.1	19.2	46.4	1.50	14.9	9.0
Parathyroid extract	83	190	Leg 7	0.391	0.206	0.250	0.132	66.7	93.0	2.32	37.2	23.8	43.9	1.42	17.5	11.2
Parathyroid extract	83	175	Leg 8	0.517	0.296	0.304	0.174	58.4	95.0	2.38	31.2	18.38	49.5	1.60	16.3	9.6
Ammonium chloride	83	176	Leg 9	0.430	0.244	0.284	0.161	68.8	94.0	2.35	33.1	21.9	47.7	1.54	16.8	11.1
Viosterol	83	175	Leg 11	0.351	0.200	0.264	0.151	76.8	85.5	2.14	32.3	24.3	44.9	1.45	17.0	12.8
Viosterol	83	164	Leg 12	0.350	0.214	0.240	0.146	70.4	79.5	1.98	33.1	22.7	41.2	1.33	17.2	11.8
Basic	118	261	Skull 2	0.788	0.218	0.470	0.180	65.6	137.5	3.44	29.3	17.4	69.0	2.22	14.0	8.8
Low calcium	118	286	Skull 3	0.673	0.236	0.385	0.135	59.8	103.5	2.61	26.9	15.4	58.8	1.96	14.7	8.7
Low calcium	118	294	Skull 4	0.573	0.195	0.380	0.130	69.2	98.5	2.46	25.9	17.2	52.6	1.70	13.8	9.0
High calcium	118	266	Skull 5	0.741	0.280	0.497	0.187	70.4	136.5	3.61	27.5	18.4	71.5	2.30	14.4	9.6
High calcium	118	256	Skull 6	0.705	0.275	0.492	0.192	71.6	136.0	3.40	27.7	19.3	75.8	2.45	15.4	10.7
Ammonium chloride	103	208	Skull 10	0.570	0.274	0.361	0.174	64.8	117.2	2.93	32.3	20.7	54.9	1.78	15.2	9.6
Basic	83	250	Skull 1	0.506	0.203	0.403	0.161	79.6	106.8	2.68	26.5	21.1	63.7	2.05	15.7	12.5
Parathyroid extract	83	190	Skull 7	0.389	0.204	0.253	0.133	65.0	96.5	2.41	38.1	24.8	39.9	1.29	17.1	10.3
Parathyroid extract	83	175	Skull 8	0.596	0.340	0.330	0.188	60.4	137.0	3.42	41.5	25.1	56.5	1.82	17.1	10.2
Ammonium chloride	83	176	Skull 9	0.382	0.218	0.270	0.153	72.6	84.8	2.12	30.3	22.2	40.9	1.32	15.1	10.7
Viosterol	83	175	Skull 11	0.329	0.190	0.234	0.134	71.0	76.0	1.90	33.1	23.1	37.5	1.21	16.3	11.4
Viosterol	83	164	Skull 12	0.380	0.232	0.234	0.143	70.7	79.5	2.00	30.55	20.9	41.0	1.32	15.8	10.8

* For convenience of reference the columns are numbered.

The long bones and the skull bones both showed a considerable decrease in the relative concentration of calcium in the ash.

The reduction in the phosphorus content of the ash was greater in the skull than in the long bones. The amounts of organic matter and water combined were greater in the skull than in the long bones.

High Calcium Experiment.—The animals which were given the high calcium diet were of about the same weight as the controls. The skeletal bones were heavier (table 5, column 5), and the amounts of ash were

TABLE 6.—*Data on Low Calcium Experiment*

Diet	Age of Rat, Days	Part Analyzed	Weights			Percentages (Control Taken as 100)		
			Ash, Gm.	Calcium, Mg.	Phosphorus, Mg.	Ash	Calcium	Phosphorus
Basic (control).....	118	Leg 2	0.371	123.5	56.8	100.0	100.0	100.0
Low calcium.....	118	Leg 3	0.320	81.0	52.3	86.6	66.6	93.8
Low calcium.....	118	Leg 4	0.315	98.0	52.6	85.0	79.3	94.3
					Average	85.8	73.0	94.0
Basic (control).....	118	Skull 2	0.470	137.5	69.0	100.0	100.0	100.0
Low calcium.....	118	Skull 3	0.385	103.5	58.8	82.0	75.3	85.4
Low calcium.....	118	Skull 4	0.380	98.5	52.6	81.0	71.5	76.2
					Average	81.5	73.4	80.8

TABLE 7.—*Data on High Calcium Experiment*

Diet	Age of Rat, Days	Part Analyzed	Weights			Percentages (Control Taken as 100)		
			Ash, Gm.	Calcium, Mg.	Phosphorus, Mg.	Ash	Calcium	Phosphorus
Basic (control).....	118	Leg 2	0.371	123.5	56.8	100.0	100.0	100.0
High calcium.....	118	Leg 5	0.431	134.5	72.4	116.0	109.0	127.0
High calcium.....	118	Leg 6	0.416	129.0	69.4	112.0	104.5	122.0
					Average	114.0	106.8	124.5
Basic (control).....	118	Skull 2	0.470	137.5	69.0	100.0	100.0	100.0
High calcium.....	118	Skull 5	0.497	136.5	71.5	105.8	99.3	103.5
High calcium.....	118	Skull 6	0.492	136.0	75.8	104.5	98.9	109.8
					Average	105.2	99.1	106.7

greater (column 7). Likewise, the absolute amounts of calcium (column 10) and of phosphorus (column 14) were greater. The increase in calcium and phosphorus in the ash was comparable to the increase in the total amount of ash.

The weights of the skulls were slightly increased in relation to the body weights (column 6); the ash weights (column 7) were increased, but the absolute amounts of calcium (column 10) and phosphorus remained relatively unchanged. However, the amounts of calcium and phosphorus, as in the long bones, were comparable to the percentage increase in ash.

Experiment with Parathyroid Extract.—The animals which were fed the diet to which parathyroid extract had been added were smaller than the controls. In animal 7 (table 5, column 7) there was a marked reduction in ash, with a slight reduction in the calcium and phosphorus content of the ash.

TABLE 8.—*Experiment with Parathyroid Extract*

Diet	Age of Rat, Days	Part Analyzed	Weights			Percentages (Control Taken as 100)		
			Ash, Gm.	Calcium, Mg.	Phosphorus, Mg.	Ash	Calcium	Phosphorus
Basic (control).....	83	Leg 1	0.309	94.0	46.4	100.0	100.0	100.0
Parathyroid extract...	83	Leg 7	0.250	93.0	43.9	81.0	99.0	95.0
Parathyroid extract...	83	Leg 8	0.304	95.0	49.5	98.5	101.0	100.5
					Average	89.8	100.0	97.8
Basic (control).....	83	Skull 1	0.403	106.8	63.7	100.0	100.0	100.0
Parathyroid extract...	83	Skull 7	0.253	96.5	39.9	62.6	90.5	88.8
Parathyroid extract...	83	Skull 8	0.330	137.0	40.9	81.7	76.7	63.2
					Average	72.1	83.6	76.0

TABLE 9.—*Experiment with Ammonium Chloride*

Diet	Age of Rat, Days	Part Analyzed	Weights			Percentages (Control Taken as 100)		
			Ash, Gm.	Calcium, Mg.	Phosphorus, Mg.	Ash	Calcium	Phosphorus
Basic (control).....	118	Leg 2	0.371	123.5	56.8	100.0	100.0	100.0
Ammonium chloride...	103	Leg 10	0.287	91.0	48.1	77.4	73.6	84.7
Basic (control).....	83	Leg 1	0.309	94.0	46.4	100.0	100.0	100.0
Ammonium chloride...	83	Leg 9	0.284	95.0	47.7	92.0	101.0	102.3
					Average	84.7	87.3	93.5
Basic (control).....	118	Skull 2	0.470	137.5	69.0	100.0	100.0	100.0
Ammonium chloride...	103	Skull 10	0.361	117.2	54.9	76.8	85.2	79.6
Basic (control).....	83	Skull 1	0.403	106.8	63.7	100.0	100.0	100.0
Ammonium chloride...	83	Skull 9	0.270	84.8	40.9	66.9	79.4	64.2
					Average	71.8	82.3	71.9

In animal 8 there was no change in the weight of the long bones or in the ash content, but there was an increase in both the calcium and the phosphorus content of the ash and an increase in the actual concentrations of these substances in the ash. However, in the skull of this animal, which was heavier than that of the control, while there was a reduction in ash, there was an apparent increase in the calcium of the ash (column 10) and a reduction in phosphorus (column 14). However, the absolute concentrations of calcium and phosphorus were reduced.

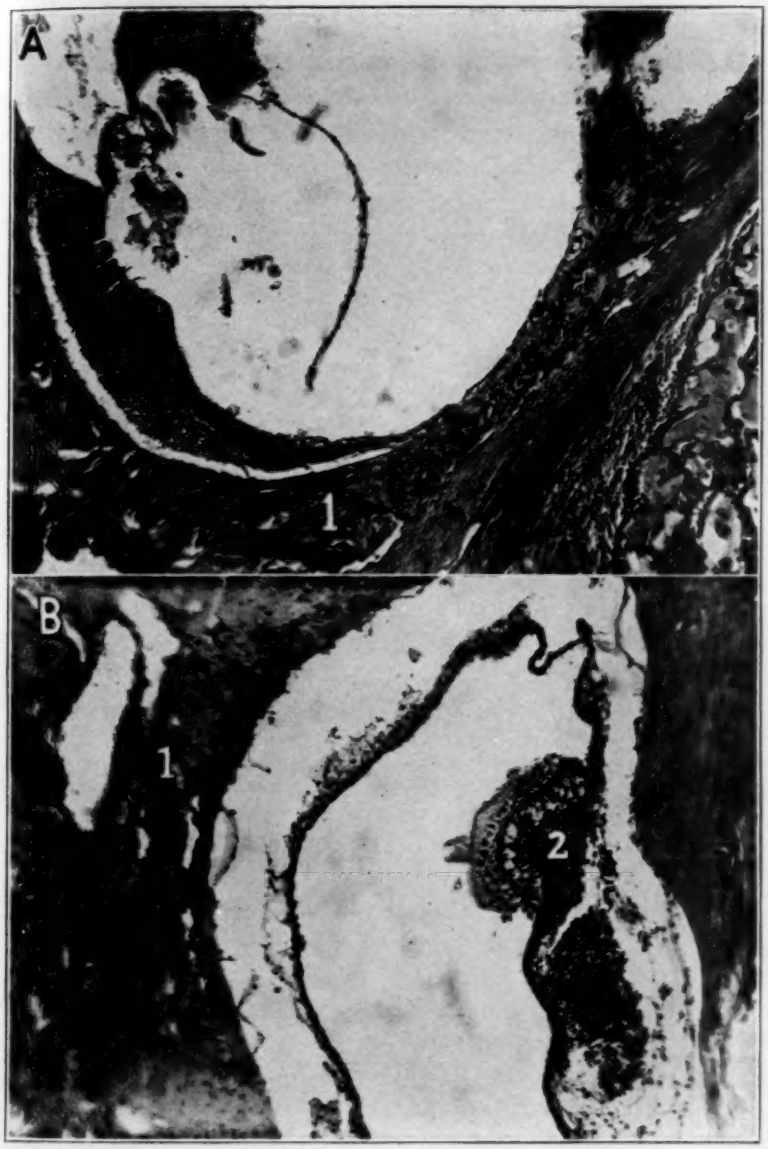

Fig. 3.—*A*, section through the cochlea of a rat which was given parathyroid extract (0.5 cc. daily). There is noted a generalized moderate resorption of the matrix, the smooth, solid matrix picture being replaced by a distorted one. Resorption cavities are scattered throughout. Osteocytes are still present in normal numbers but do not show the normal arrangement, owing to the disturbed condition of the matrix. *1* indicates the otic capsule. *B*, section through an ampulla of a rat which was given parathyroid extract (0.5 cc. daily). *1* indicates the capsule, which shows marked halisteresis. Cavities have appeared. Osteocytes are disarranged. The crista ampullaris (*2*) is normal. This alteration of the vestibular labyrinth, greater than that noted in the cochlear region, is unexpected. The vestibular labyrinth, phylogenetically much older than the cochlear, is usually the more resistant to change.

As in the experiments with ammonium chloride, care must be taken in the interpretation of these apparently discordant results. The usual findings in rats after large doses of parathyroid extract are lessened

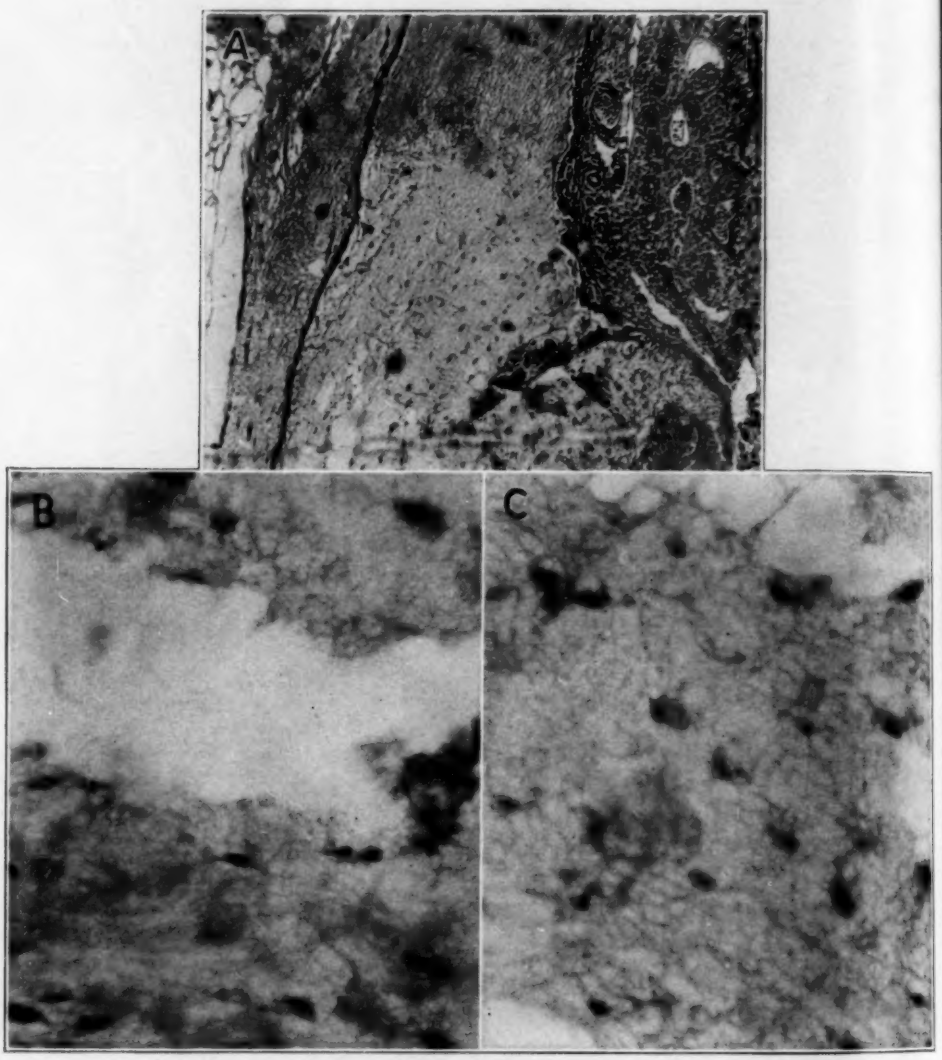

Fig. 4.—Section through the skull, away from the ear, of a rat which was given parathyroid extract (0.5 cc. daily). There is present much more marked halisteresis than in the aural region. *A* shows marked halisteresis of the matrix, shading off into less affected bone. In places, matrix and osteocytes have entirely disappeared. *B* and *C* show high magnifications of the most affected areas in *A*. Some of the osteocytes are wandering off as fibroblasts. This greater involvement of bone, elsewhere than in the otic capsule, caused us to reject parathyroid as a decalcifying agent in the final half of the research.

appetite, decreased rate of growth and smaller bones, which contain less ash and less of calcium and phosphorus.

Ammonium Chloride Experiment.—The weights of the animals to the diet of which ammonium chloride was added were less (table 5, column 3) than those of the controls, and the weight of the bone and ash decreased (column 5 and column 7). The absolute amounts of calcium and phosphorus were decreased (10 and 14). The reduction in calcium was somewhat greater than the decrease in ash. The phosphorus was relatively increased.

In the younger animal (83 days), the body was smaller, but the weights of the bones (column 5), the ash contents (column 7) and the absolute amounts of calcium (column 10) and phosphorus (column 14) were increased. The increase in calcium was greater than the increase in ash.

TABLE 10.—*Experiment with Viosterol*

Diet	Age of Rat, Days	Part Analyzed	Weights			Percentages (Control Taken as 100)		
			Ash, Gm.	Calcium, Mg.	Phosphorus, Mg.	Ash	Calcium	Phosphorus
Basic (control).....	83	Leg 1	0.309	94.0	46.4	100.0	100.0	100.0
Viosterol.....	83	Leg 11	0.264	85.5	44.9	85.5	91.0	98.0
Viosterol.....	83	Leg 12	0.240	79.5	41.2	77.6	84.5	89.0
					Average	81.6	87.8	93.5
Basic (control).....	83	Skull 1	0.403	106.8	63.7	100.0	100.0	100.0
Viosterol.....	83	Skull 11	0.234	76.0	37.5	58.0	71.0	58.8
Viosterol.....	83	Skull 12	0.234	79.5	41.0	58.0	74.5	64.2
					Average	58.0	72.8	61.5

In the skulls of the animals which were given ammonium chloride, a marked difference of reaction occurred. Both animals had smaller skulls (column 5), which showed less ash (column 7) and a marked decrease in amounts of calcium (column 10) and phosphorus (column 14). However, while the reduction in phosphorus was due to the degree of reduction in ash, there was relatively a greater concentration of calcium in the skull in proportion to the ash than in the control animals. Phosphates, water and organic material were lost in greater degree than was calcium.

Experiment with Viosterol.—The animals were small (table 5, column 3), the ash reduced and the amounts of calcium and phosphorus reduced (columns 7, 10, 14). However, the reduction in concentration was mainly in the calcium, which was approximately proportional to the reduction in the ash.

The skulls were small (column 5), the ash reduced (column 7) and the amounts of calcium and phosphorus reduced (columns 10 and 14). However, the concentration of calcium was reduced to a lesser degree than was that of the phosphorus, which was reduced in proportion to the reduction of ash.

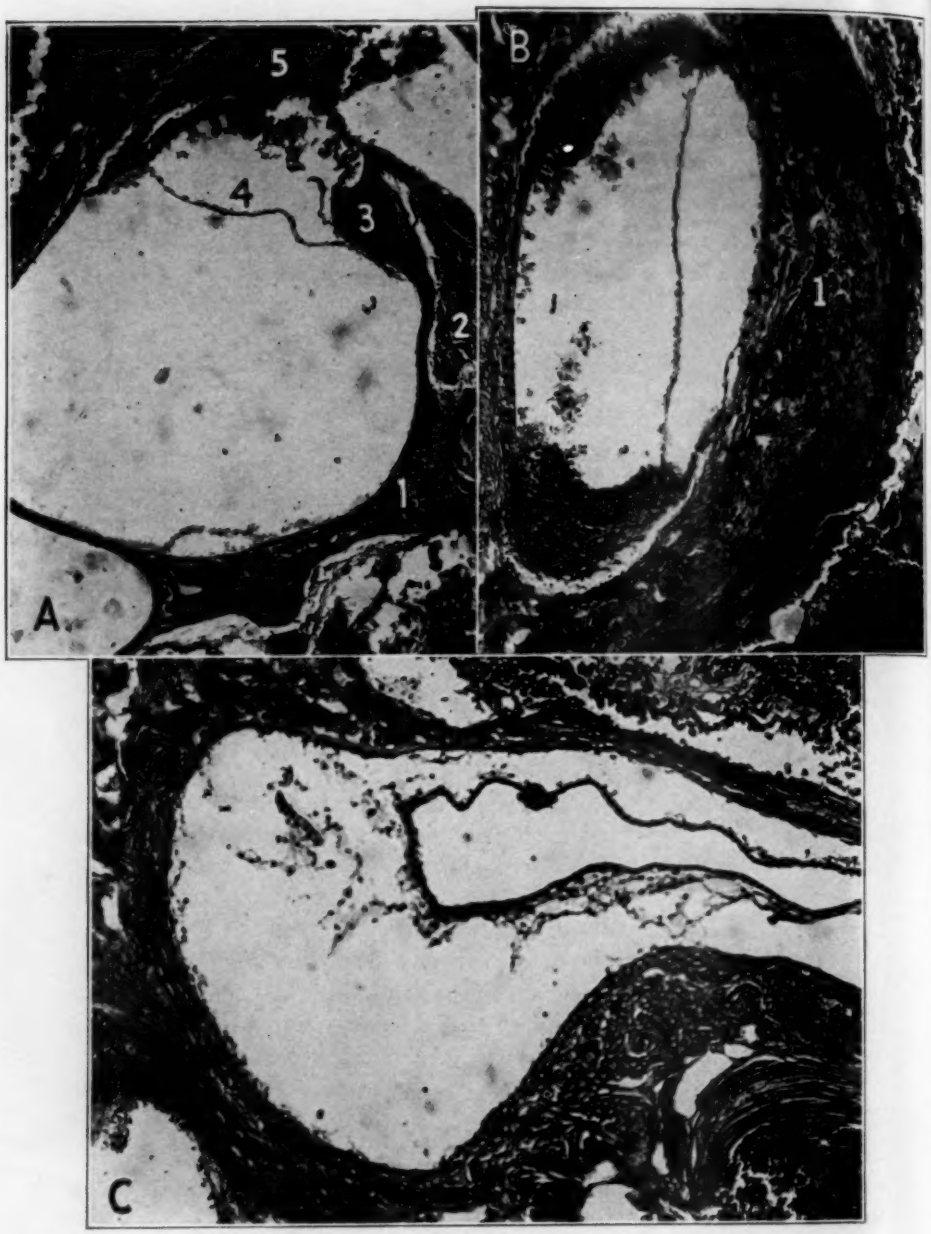

Figure 5

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As with parathyroid extract, the toxic effects of viosterol must be evaluated with care. In the long bones the reduction of ash is mainly an effect of withdrawal of calcium; in the membrane bones, mainly of a withdrawal of phosphorus. Both of the animals to which viosterol was given were immature.

EFFECTS OF DECALCIFYING AGENTS ON THE OTIC CAPSULE

There was observed no difference in the appearance of the capsule of the labyrinth between the rats which were fed the basic diet and those which were given the low and the high calcium diets.

Some interesting facts concerning the effects of parathyroid extract on rats are gleaned from the work of Burrows.[2]

Three units daily (0.15 cc.) of parathyroid extract (prepared according to Collip's method) resulted in slowing of growth. More than 3 units caused rapid loss of weight and death in a few days. Hypoplasia of parathyroid tissue followed the larger dose. The first visible effect on long bones was a decrease in trabeculae, with increased density of compact bone. These changes were noted in both nonparathyroidectomized and partially parathyroidectomized rats. No gross change in the shape of the skull followed injection of the extract. The skull and the leg bones were small, both in proportion to the diminished size of the whole animal. In the skull the compact layer became denser, while the diploe showed rarefaction. No difference was noted in the response of cartilage and membrane bone. Microscopically, resorption was seen, with a decrease in osteoblasts, then replacement with fibrous tissue. Osteoclasts had increased.

2. Burrows, R. B.: Variation Produced in Bones of Growing Rats by Parathyroid Extracts, Am. J. Anat. **62**:237-289, 1938.

EXPLANATION OF FIGURE 5

Fig. 5.—*A*, section through the cochlea of a rat which had had ammonium chloride (250 mg. per day) added to the basic diet. *1* indicates the capsule, which shows a marked decrease in osteocytes, a general thinning of matrix and numerous areas of resorption. Gray-blue staining of bone with hematoxylin-eosin, instead of the normal red, was noted. *2* indicates the spiral ganglion; *3*, the limbus laminae spiralis; 4, the vestibular membrane, and 5 the ligamentum spirale cochleae. *B*, section through the cochlea of a rat which had had ammonium chloride (250 mg. per day) added to the basic diet. *1* indicates the capsule, which shows irregularity of mobilizing osteocytes, general thinning of matrix and areas of resorption. The bone stained gray-blue with hematoxylin-eosin, instead of normal red. *C*, section through the vestibular capsule of a rat which had had ammonium chloride (250 mg. per day) added to the basic diet. Halisteresis has produced a bizarre appearance of the bone, marked loss of osteocytes, thinning of matrix and resorption areas. The staining of the bone with hematoxylin-eosin showed a pale washed-out blue instead of the normal red.

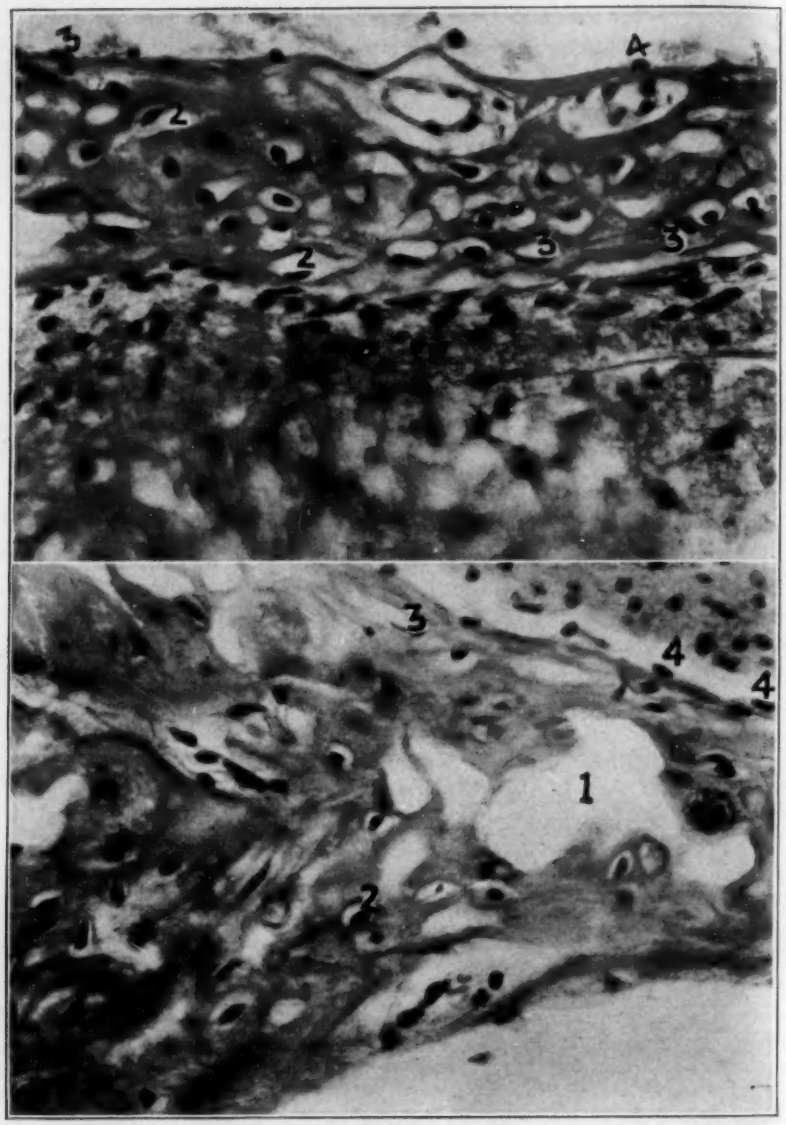

Fig. 6.—High magnification of the cochlear capsule of a rat which had had ammonium chloride (250 mg. per day) added to the basic diet. Note (*2*) the peculiar resorption of bone around the osteocytes, (*1*) large areas of complete resorption, (*3*) fibroblasts formed from osteocytes and generalized thinning of matrix. Some of the osteocytes (*4*) have wandered, as fibroblasts, completely away from the bone.

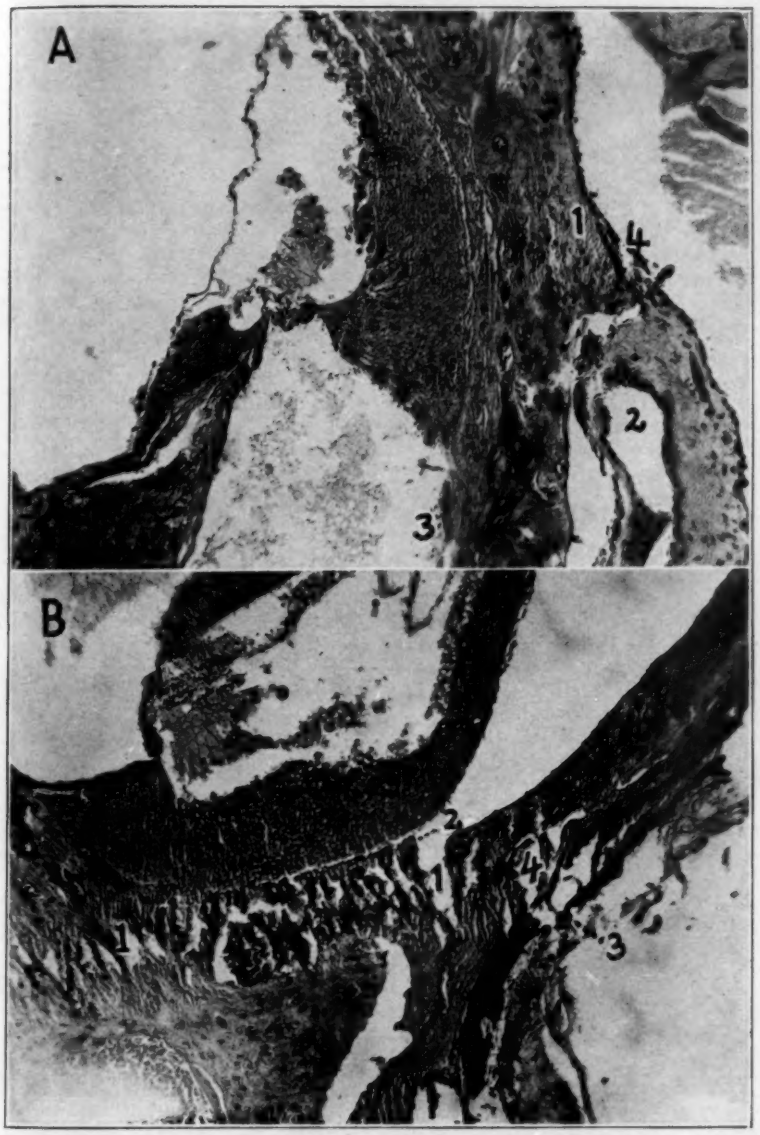

Fig. 7.—*A*, section through the cochlea of a rat which had had viosterol (34,000 units daily) added to the basic diet. The capsule (*1*) shows disarranged osteocytes from beginning mobilization. The matrix is rapidly disintegrating from loss of calcium and phosphorus. *2* indicates resorption area. The endosteum (*3*) and periosteum (*4*) are disturbed in the process of halisteresis. All three layers of the capsule are affected, but a somewhat greater change is noted in the periosteal and endosteal layers. *B*, section through the cochlea of a rat which had had viosterol (34,000 units daily) added to the basic diet. The labyrinthine capsule is in shreds from marked halisteresis. Osteocytes have to a great extent disappeared. *1* indicates the markedly disintegrated capsule; *2*, the endosteum destroyed; *3*, the periosteum destroyed, and *4*, resorption area.

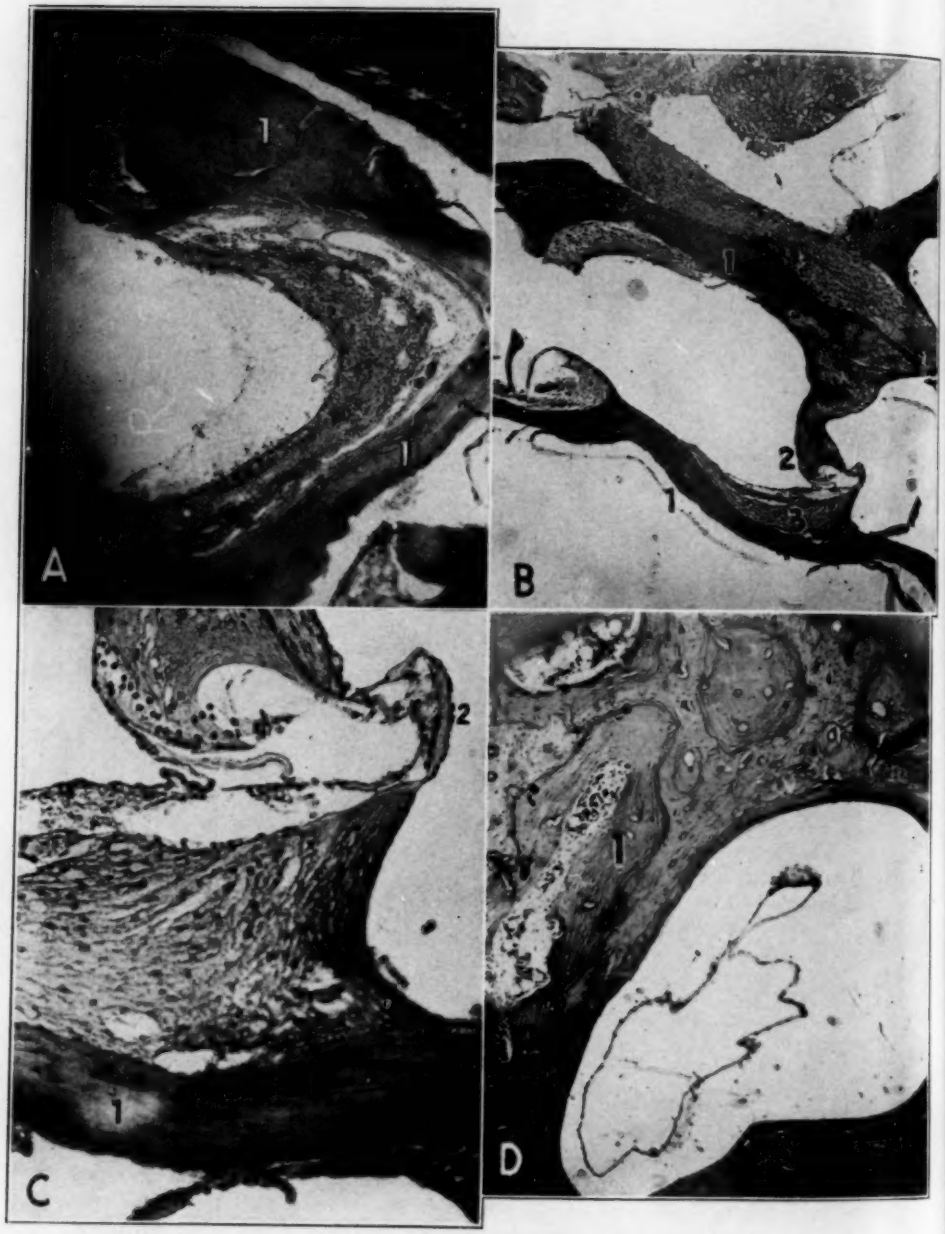

Figure 8

(See legend on opposite page)

Continued injection of parathyroid extract caused a reversal of these processes. Osteoclasts disappeared, osteoblasts reappeared, fibrous tissue diminished, and hypercalcification ensued (marble bone).

The interesting transition from the picture of osteitis fibrosa to that of marble bone may be due to an antibody-like substance, which inhibits decalcification. Hypercalcification is then possibly checked by another substance in the blood, acting against the antibody-like factor.

After study of the various methods of decalcification, the conclusion was reached that ammonium chloride would probably serve best in the second half of the research, in which halisteresis was produced in the otic capsule and the process was then reversed. The selection of this agent resulted from the fact that more marked changes occurred in the otic capsule from its use than elsewhere; in fact, a selective effect on the aural capsule may be claimed.

REVERSAL OF HALISTERESIS

Though rats synthesize vitamin C, it was thought best to include in the basic diet 1 ounce (28 cc.) of orange juice daily (freshly prepared), as the gelling of collagen in recalcification requires a plentiful amount of this vitamin.

Ten rats (Wistar stock, white, all females, all 45 days old, of the same strain as those used in the first half of the experiment) were utilized.

EXPLANATION OF FIGURE 8

Fig. 8.—*A*, section through the cochlea of rat 5, which was given the basic diet plus 250 mg. of ammonium chloride daily and was killed on the twenty-seventh day. The otic capsule (*1*) shows almost no bone lamellae; osteocytes have decreased in number. There is a thinning of matrix. The bone has a washed-out appearance and stained gray-blue with hematoxylin-eosin. *B*, section through the cochlea of rat 6, which was given the basic diet plus 250 mg. of ammonium chloride daily for twenty-seven days, showing the mottled washed-out appearance of the capsule, decrease in the number of osteocytes and thinning of the matrix. *1* indicates the capsule undergoing halisteresis from acidosis; *2*, the limbus laminae spiralis, and *3*, the ligamentum spirale cochleae. *C*, section through the cochlea of rat 6, showing evidence of halisteresis of the capsule. The capsule (*1*) stained gray-blue and showed an absence of normal detail. *2* indicates the basilar membrane. *D*, section through the labyrinthine capsule of rat 3, which had been given 250 mg. of ammonium chloride daily for twenty days, then a basic-high calcium diet for forty-two days. The halisteresis from ammonium chloride has almost entirely disappeared, and normal lamellated matrix, numerous osteocytes and normal red-staining matrix are seen. Enlarged lacunas persist.

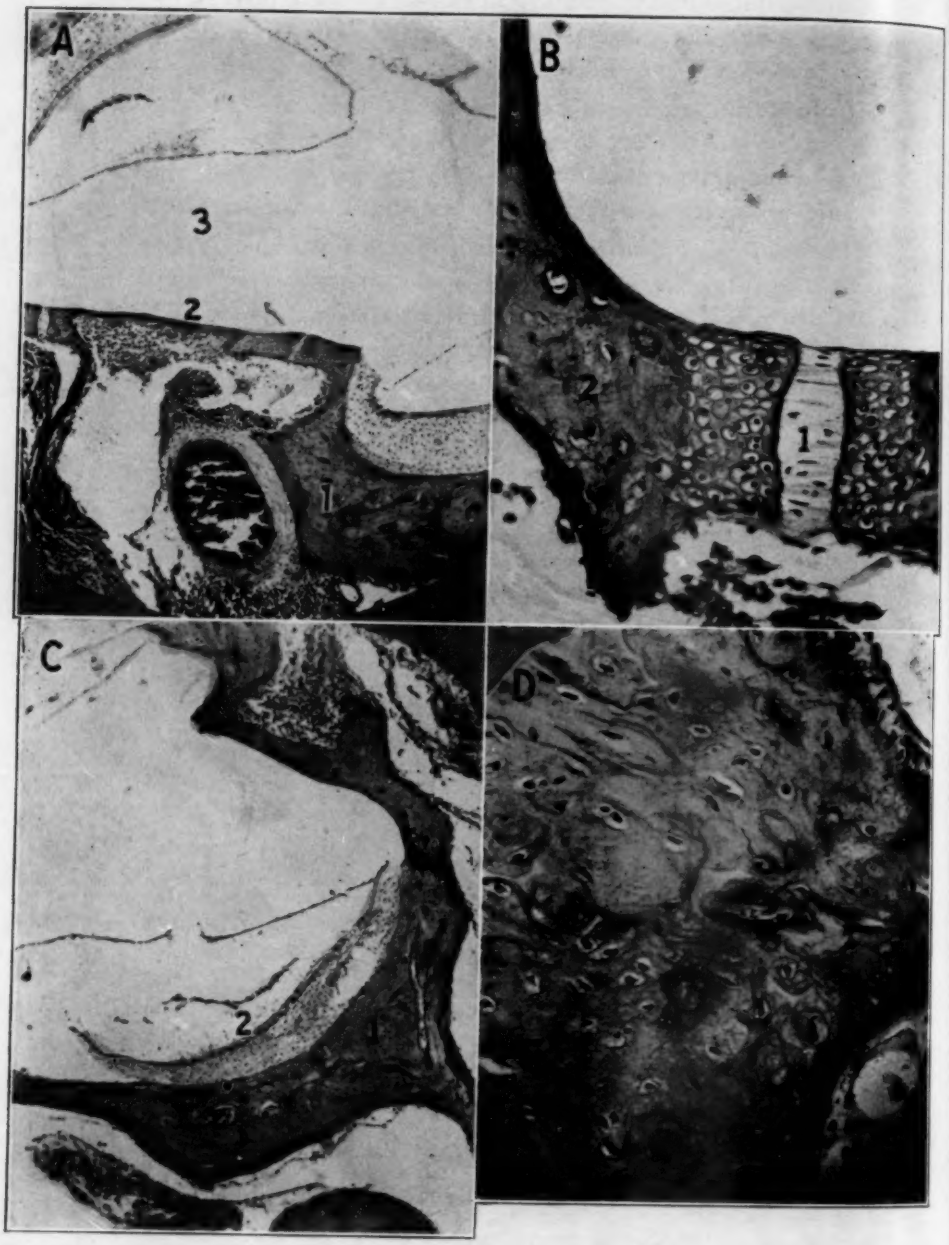

Figure 9

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The experiment was started May 10, 1938, with all 10 rats on the basic diet for one week. On May 18, rats 1, 2, 3, 4, 5 and 6 were placed on the basic diet plus 250 mg. daily of ammonium chloride. On May 31 it was noted that a yellowish brown discoloration and loss of hair were developing in rats 1, 2, 3, 4, 5 and 6. Rats 7, 8, 9 and 10 remained perfectly white. Rat 6 lost weight rapidly. Rats 1, 2, 3, 4 and 5, although discolored, seemed to be well otherwise but did not gain as rapidly in weight as the control rats, 7, 8, 9 and 10. On June 4, rats 5 and 6 were put to death. On June 10, rats 7 and 8 were killed. On June 7, rats 1, 2, 3, 4, 9 and 10 were placed on a high calcium diet free

TABLE 11.—*Weights (Gm.) of Rats in Second Half of Experiment (the Production and Reversal of Halisteresis)*

Date	Rat 1	Rat 2	Rat 3	Rat 4	Rat 5	Rat 6	Rat 7	Rat 8	Rat 9	Rat 10
5/10/38	76	68	75	70	59	71	70	70	62	58
5/14/38	75	68	74	68	59	71	70	71	61	58
5/16/38	76	68	76	69	61	72	71	72	62	59
5/18/38	76	69	77	70	61	72	72	72	63	60
5/20/38	77	71	77	71	62	73	72	72	63	61
5/28/38	80	81	82	81	68	76	84	88	84	85
5/31/38	81	83	83	83	74	68	85	90	86	88
6/ 2/38	83	83	94	83	87	54	87	92	98	92
6/ 4/38	109	101	120	99	94	72	104	108	118	105
					killed	killed				
6/ 7/38	136	122	144	120	...	...	127	130	138	119
6/10/38	139	130	148	126	...	...	128	130	142	127
							killed	killed		
6/15/38	143	138	151	132	...	...	...	...	149	135
6/27/38	176	148	102	155	...	...	...	...	173	151
6/29/38	171	148	151	154	...	...	...	...	170	150
	killed									
7/ 4/38	...	149	150	151	...	...	...	...	165	153
7/11/38	...	151	151	152	...	...	...	...	158	151
7/19/38	...	153	152	150	...	...	...	...	150	150
Remaining rats killed										
Average daily gain, Gm.....	1.9	1.3	1.2	1.3	1.5	0.04	2	2.06	1.7	1.4

of ammonium chloride. On June 29, rat 1 was killed. On July 19, all the remaining rats were sacrificed.

EXPLANATION OF FIGURE 9

Fig. 9.—*A*, section through oval window region of rat 4, which had been given 250 mg. of ammonium chloride for twenty days, then a basic–high calcium diet for forty-six days. The halisteresis produced by the ammonium chloride has almost entirely disappeared, and the otic capsule is seen to be normal except for slightly enlarged lacunas. The region shown is that of the stapes and oval window; *1* indicates the capsule; *2*, the footplate of the stapes, and *3*, the cisterna. *B*, high magnification of the annular ligament area in *A*. Note the solid matrix. *1* indicates the annular ligament; *2*, the capsule of the labyrinth. Lacunar enlargement is the only remaining abnormality. *C*, section through a macula acustica (*2*). The capsule of the labyrinth (*1*) is normal except for enlarged lacunas. Halisteresis has been reversed. *D*, high magnification of the aural capsule in *C*. Halisteresis has been reversed except for persisting enlargement of the lacunas.

The average daily gain of the rats which were given ammonium chloride was 1.15 Gm., while the control rats, on the basic diet, gained daily 1.79 gm.—much less difference than one would have anticipated. in view of the profoundly altered appearance of the rats which were given ammonium chloride, particularly number 6.

SUMMARY

Decalcification of the aural capsule was produced in Wistar white rats with parathyroid extract, viosterol and ammonium chloride. The most marked halisteresis of the aural capsule as compared with bone changes elsewhere resulted from the administration of ammonium chloride.

With a second group of animals, ammonium chloride was used to produce decalcification, after which a reversal of the process (osseous reconstruction) was accomplished by removing ammonium chloride from the diet and placing the rats on a high calcium diet.

CONCLUSIONS

1. Clinically it seems possible to inhibit the enlargement of an otosclerotic focus with adequate amounts of calcium, phosphorus and vitamins.

2. The explanation of this inhibition would seem to be the reconstruction of capsular bone in the decalcifying areas around the dystrophic focus.

Dr. David M. Greenberg assisted in the study, and Eleanor Steins prepared the manuscript and assisted with the photomicrography.

QUANTITATIVE EFFECTS OF PHYSICAL AND CHEMICAL AGENTS ON THE ERECTILE TISSUE RESPONSE IN THE NOSE

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In previous communications [1] a new and simple manometric method was described for the accurate measurement of nasal aerodynamics. It was shown that by this method one could determine the comparative shrinkage efficiency of vasoconstrictor drugs commonly used in the treatment of nasal obstruction.[2] Before investigating the specific action of these drugs as administered by means of nasal sprays, it appeared essential to evaluate the physiologic effects of certain factors, such as (*a*) air pressure and (*b*) the tonicity of a solution, on the erectile tissue response of the inferior turbinate. The present study deals with an investigation of these factors.

METHOD OF STUDY

The apparatus (fig. 1) used was made up of a nasal catheter (*F*) with glass olive tips of suitable size; a standard flowmeter, to measure the volume of air flow (liters per minute); a convenient source of suction (*V*) and a series of two manometers, inclined (*E*) [3] and vertical (*V* [1]), to measure the resistance to nasal air flow (millimeters of water).

The procedure consisted of a clinical examination of the nose and throat followed by preliminary instructions both as to the type of mouth breathing required and the avoidance of swallowing during the test. Nasal secretions were either aspirated or blown out by the patient. The flowmeter was set in operation by turning on the suction (fig. 1, *V*) and tightening down the screw clamp (*B*) until the manometer (*D*) registered the necessary air flow in liters per minute. The

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This study was aided by a grant from the Charlton Research Fund of the Tufts College Medical School.

1. (*a*) Sternstein, H. J., and Schur, M. O.: Quantitative Study of Nasal Obstruction: A New Method, Arch. Otolaryng. **23**:475 (April) 1936. (*b*) Sternstein, H. J.: Nasal Obstruction in the Adult: A Quantitative Study, ibid. **25**:442 (April) 1937.

2. Tourangeau, J. J., and Drinker, P.: The Dust Filtering Efficiency of the Human Nose, J. Indust. Hyg. **19**:53 (Jan.) 1937.

3. Incline ¼ inch per inch (0.7 cm. per 2.6 cm.) length of scale.

sliding scale on the inclined manometer was adjusted to the zero reading, to correct for the resistance factor produced by the nasal catheter. With the tongue held as shown in figure 1 and the subject breathing calmly through the mouth, an olive tip of suitable size was inserted gently into the nostril and a measured volume of air drawn in through the open mouth, postnasal space and nasal airway. The resistance encountered by the flow of air through the nasal passage was read from the inclined or vertical manometer.

Comment.—The use of calm voluntary mouth breathing with the tongue held as shown in figure 1 served to prevent, within physiologic

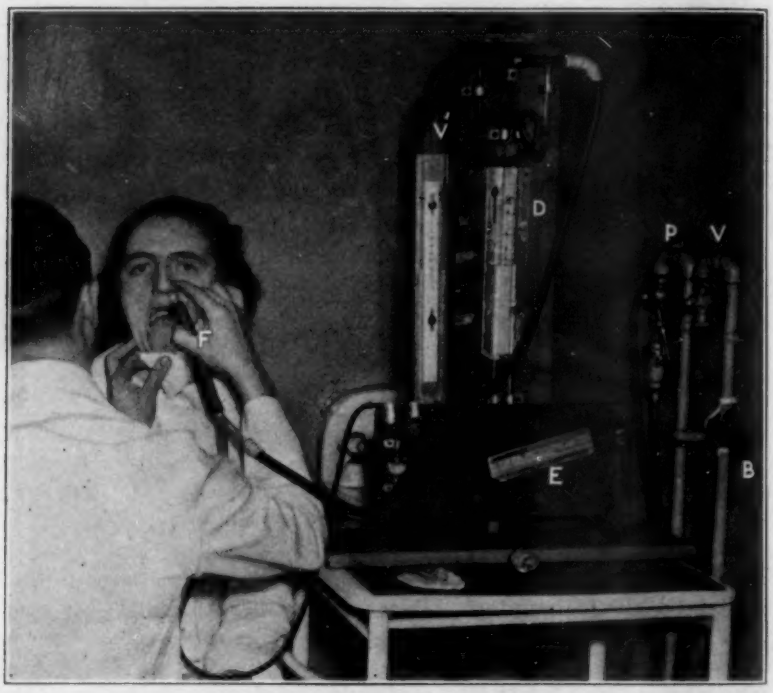

Fig. 1.—Photograph of the apparatus, showing the technic and the relative positions of the nasal catheter and the mouth during the test. The vertical manometer (V^1) measures the resistance (millimeters of water) beyond the range of the inclined manometer scale.

limits, muscular contractions of the oropharynx and the alae nasi as well as respiratory variations in the nasal tract as experimental sources of error otherwise present in direct manometric methods.[4] In earlier experi-

4. Uddstromer, M.: Nasal Respiration: A Critical Survey of Some Current Physiological and Clinical Aspects on the Respiratory Mechanism with a Description of a New Method of Diagnosis, Acta oto-laryng., 1940, supp. 42, p. 29.

ments [1b] it was found that air flow through the nose, as regulated by the degree of swelling of the erectile tissue of the inferior turbinate, is governed by the same physical laws that apply to the passage of fluids through any constricted orifice. Since it was observed that simultaneous measurements of both sides of the nose determined only the physiologic resistance of the more patent side without giving any accurate knowledge of the actual degree of obstruction present, each nasal airway was separately measured for its dynamic function.

The physical and chemical agents employed as experimental stimuli were (*a*) positive air pressure (15 lb. [6.8 Kg.]) released into each nostril from a DeVilbiss nozzle for one to two seconds and (*b*) saline solutions of 0.5 per cent, 0.8 per cent, 1 per cent and 5 per cent concentrations sprayed on the nasal mucous membrane. About 0.6 cc. of solution, approximately 6 drops, was used for each nostril.

Resistance measurements obtained before and after stimulation were recorded at an air flow of 12 liters per minute. Earlier experiments were based on measurements taken at air flows of 4, 8, 12 and 16 liters per minute. In the experiments to be reported presently an easily tolerated air flow of 12 liters per minute was routinely used, since it was found [5] to correspond approximately with the volume of air breathed per minute during a state of moderate physical activity. Consecutive readings were taken at minute intervals for a period varying from three to five minutes, and this procedure was repeated every twenty minutes over a total period of one-half to two hours. The measurements were averaged and tabulated according to the subjectively patent and the subjectively blocked nasal passages. Corrections were made for the alcohol manometer readings to the conventional millimeters of water for the degree of inclined gage (*E*) used.

Experiments were conducted at average room temperatures, with average humidity (20 C., 760 mm. of mercury).

CLINICAL STUDIES

The clinical studies were conducted with subjects who were selected from the hospital personnel and from the nose and throat clinic. The patients were classified according to their chief complaint into a blockage and a nonblockage group. In the latter group were included those who presented physical signs of obstruction, such as enlargement or swelling of the inferior turbinates, septal deviations or spurs, yet did not complain of nasal blockage. This condition was designated as functional blockage. The blockage group was represented by the patients with acute head cold, acute vasomotor disturbance and chronic inflammatory and polypoid sinusitis. Thus 42 patients were observed during the periods

5. Henderson, Y., and Haggard, H. W.: Noxious Gases and the Principles of Respiration Influencing Their Action, American Chemical Society Monograph Series, New York, The Chemical Catalog Company, 1927, no. 35, p. 30.

of quiescence and activity of their blockage symptoms and were arranged as follows:

Nonblockage Group	Patients
Normal	6
Vasomotor rhinitis (quiescent)	7
Chronic catarrhal ethmoiditis (quiescent)	4
Chronic hypertrophic rhinitis (quiescent)	3
Total	20

Blockage Group	Patients
Vasomotor rhinitis (active)	12
Vasomotor polypoid ethmoiditis (active)	3
Chronic catarrhal ethmoiditis (active)	2
Chronic hypertrophic rhinitis (active)	5
Total	22

In the total group of 42 patients there were 24 females and 18 males. The ages varied from 18 to 55 years, with an average of 38.4 years. The patients were under observation over a period of two to seven months. With the exception of the normal subjects in the nonblockage classification, all gave a previous history of intermittent and alternating obstruction extending over a variable period of one to fifteen years.

The clinical findings, in both the nonblockage and the blockage group, were tabulated according to the structural appearance of the septum, the erectile tissue of the inferior turbinate and the roominess of the nasal airways.

In the nonblockage group septal abnormalities consisted of deviation and spurs. The middle and inferior turbinates varied in their gross appearance.

In 86.2 per cent of the patients in the blockage group a direct correlation was observed between the septal deviation and the existing type of blockage.

EXPERIMENTAL STUDIES

In the two groups 142 experimental studies were conducted. They are classified as follows:

Nonblockage Group	Patients	Experiments
Normal	6	11
Vasomotor rhinitis (quiescent)	7	28
Chronic catarrhal ethmoiditis (quiescent)	4	8
Chronic hypertrophic rhinitis (quiescent)	3	13
Total		60

Blockage Group		
Vasomotor rhinitis (active)	12	44
Vasomotor polypoid ethmoiditis (active)	3	
Chronic catarrhal ethmoiditis (active)	2	19
Chronic hypertrophic rhinitis (active)	5	19
Total		82

A total of 580 resistance measurements were tabulated according to subjective nonobstruction and subjective obstruction of the separate nasal airways. An average interval of forty minutes before and after stimula-

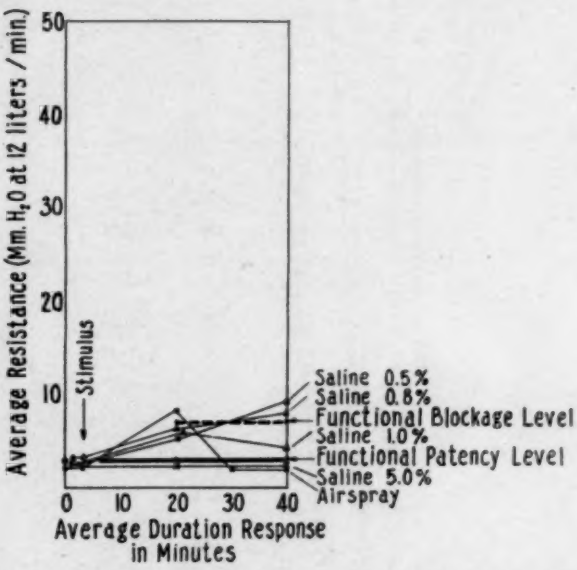

Fig. 2.—The erectile tissue response in a nonobstructed nose before and after stimulation. The heavy black line represents the average normal resistance, designated as the "functional patency level." The heavy dotted line represents the minimum average blockage resistance, designated as the "functional blockage level."

tion served as a basis for comparison of the experimental effects produced. The measurements were also correlated as to seasonal changes.

Nonblockage Group.—In this group the measurements were found to range between averages of 1 and 4 mm., with a median average of 3.25 mm. designated as the "functional patency level" (fig. 2). In the patients with functional blockage, i. e., when the nose appeared to be obstructed and yet the subject was unaware of nasal blockage, the resistance ranged from averages of 4 to 9 mm., with a median average of 6.5 mm. designated as the "functional blockage level." After stimulation

of the nasal membranes with air under pressure (15 pounds [6.8 Kg.]) there was a gradual increase in resistance within twenty minutes to a point slightly above the blockage level. This was followed by a corresponding decrease during the next twenty minutes. Spraying with 0.5 per cent and 0.8 per cent produced a gradual increase in resistance to a point slightly above the blockage level within thirty minutes. The

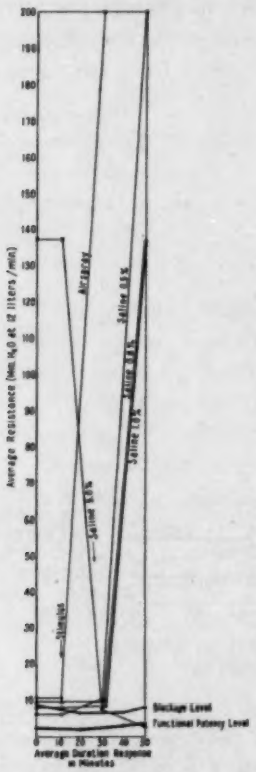

Fig. 3.—The response of the erectile tissue before and after stimulation in a nose with obstruction due to chronic hypertrophic rhinitis. Note the opposite reactions produced with isotonic (0.8 per cent) and hypertonic (5 per cent) saline solutions.

hypertonic saline solution (1 per cent and 5 per cent) produced no measurable increase in the resistance measurements.

Blockage Group.—In this group the resistance of the nonobstructed side was found to be a median average of 3.75 mm. almost identical with that of the nonblockage group except for a negligible statistical variation. In the subacute, or chronic, type of obstruction the resistance measurements of the obstructed side ranged between averages of 6.5

and 10 mm. with a median average of 8.25 mm. as the minimum abnormal blockage level. The resistance averages for the acute clinical conditions were found to be exceedingly greater and varied, according to the state of clinical activity present, from 10 to 400 mm. (table). After experimental stimulation the median average resistance for the patent, or nonobstructed, side was not appreciably altered (3.75 to 4 mm.). In the patients with chronic hypertrophic rhinitis (fig. 3) the experimental agents, with the exception of a 5 per cent saline solution, provoked a marked increase in resistance as compared with the response

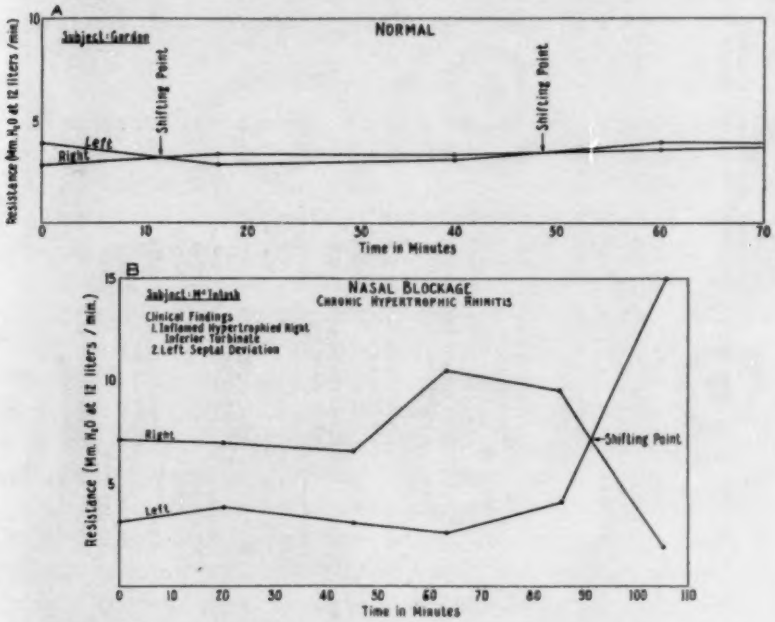

Fig. 4.—Plotted curves to show (*A*) the alternating reflex activity of the erectile tissue in a nonobstructed nose, as indicated by the "shifting points," and (*B*), the alternating functional variations of the swelling of the erectile tissue in an obstructed nose.

elicited in patients with vasomotor rhinitis and in those with chronic catarrhal ethmoiditis. In the patients with vasomotor rhinitis, an isotonic saline solution (0.8 per cent) produced a rise in resistance and nasal obstruction twenty minutes after the stimulus was applied. In general, in both the patients with vasomotor rhinitis and those with chronic catarrhal ethmoiditis blockage reactions to air pressure of approximately 15 pounds (6.8 Kg.) and to the electrolytes ranging in tonicity from 0.5 per cent to 1 per cent were induced within a total range of 6.5 to 20 mm. However, a 5 per cent hypertonic saline

A Clinical Comparison of the Average Resistance Measurements (Millimeters of Water at 12 Liters per Minute) During Nasal Patency and Blockage

Age, Yr.; Sex; Weight, Lb.	Subject	Clinical Diagnosis	Duration of Blockage	Type of Blockage	Average Blockage Resistance, Mm. H_2O	Average Nonblockage Resistance, Mm. H_2O	Experiments	Period of Observation	Deviation from Normal *	Comment
53 F 183	Cooper.....	Acute rhinitis Subacute rhinitis	7th day 21st day	Intermittent and alternating	14.5 6.9	4.0	13	4 mo.	+0.75	
24 M 194	McIntosh....	Chronic hypertrophic rhinitis Deviated septum Acute rhinitis	5 yr. 7th day	Constant and alternating	8.50 22.50	3.25	23	5 mo.	0.0	
30 M 139	Olson.....	Chronic hypertrophic rhinitis Deviated septum	.....	Intermittent and alternating	7.75	2.0	6	2 mo.	—1.25	
18 M 145	Starr.....	Chronic hypertrophic rhinitis Deviated septum Acute rhinitis Acute rhinitis †	2 yr. 7th day 1 day	Intermittent and alternating	6.0 6.75 8.50	3.25	16	5 mo.	0.0	
50 M 128	Monsour.....	Vasomotor rhinitis	1 yr.	Intermittent and alternating	9.0	2.50	22	7 mo.	—0.75	Blockage more marked on left
22 F 130	Kaplinsky....	Vasomotor rhinitis Acute rhinitis	5 mo. 2d day	Intermittent and alternating	7.0 >400 #	3.75	9	4 mo.	+0.25	Blockage more marked on left
39 F 137	Dantona.....	Chronic polypoid ethmoiditis (Vasomotor) Acute rhinitis	5 yr. 7th day 14th day 9th week	Intermittent and alternating	9.50 12.25 7.00 16.50 §	3.25	11 ..	4 mo.	0.0 ...	
45 M 156	Detullio.....	Chronic polypoid ethmoiditis Chronic nasopharyngitis Acute rhinitis	20 yr. 7th day 17th day 6th week	Intermittent and alternating	8.25 5.00 9.00 15.00 ¶	4.5	10 ..	4 mo.	+1.25	Blockage more marked on right
35 F 135	Straymann..	Allergic rhinitis Acute rhinitis Acute rhinitis ‡	5 yr. 2d day 1st day	Intermittent and alternating	10.75 54.0 135.0	3.75	14 ..	6 mo.	+0.50	2d acute exacerbation
52 F 145	Katzeff.....	Vasomotor rhinitis	1 yr.	Intermittent and alternating	5.75	4.0	5	2 mo.	+0.75	

* Normal = 3.25 mm. of water.
† Three weeks later.
# Off the vertical scale.
§ Not improved with treatment; polypectomy advised.
¶ Not improved with treatment; polypectomy advised.
‡ One month later.

solution was found invariably to produce a vasoconstrictor or shrinkage response, as indicated by the rapid decrease in resistance.

The acute type of blockage shown in the table was difficult to study experimentally, because of the discomfort present. The patients, however, were observed during the subacute or chronic stages. It is of interest that the results of clinical treatment could be followed easily with the measurements obtained.

A comparison of the reflex activity of the erectile tissue of the inferior turbinate in a normal and in a blocked nose is demonstrated in the plotted resistance curves shown in figure 4.

Seasonal variations had no bearing on the results of the experiments.

COMMENT

The reaction of the erectile tissue of the inferior turbinate to physical stimulation was markedly different in the nonblockage and blockage groups. The weak response in the former as compared with the marked intensity of reaction in the latter group clearly indicated the difference in degree of parasympathetic activity.

The most surprising result of the investigation was the completely unexpected effect of isotonic saline solution (0.8 per cent) on the swelling of the erectile tissue in an obstructed nose, as manifested by the apparent increase in resistance. The general belief that isotonicity constitutes an ideal condition for the use of vasoconstrictor drugs apparently was not supported by the experimental evidence. It was noted instead that hypertonic solutions (5 per cent) exerted an effective shrinkage action on the edematous nasal mucosa. It is difficult to interpret the mechanism of these reactions except possibly through the investigations of Buhrmester and Wenner,[6] who observed an altered permeability of tissue in the presence of nasal polpi and allergic disorders.

SUMMARY

An experimental investigation was conducted with 42 subjects, to determine the quantitative effects of (*a*) positive air pressure (15 pounds [6.8 Kg.]) and (*b*) solutions of sodium chloride ranging in tonicity from 0.5 to 5 per cent on the reflex behavior of the erectile tissue of the inferior turbinate in the nonblocked and in the blocked nose.

The rate and degree of vasodilatation and vasoconstriction in each nasal airway were measured by means of a manometric method in terms of resistance (millimeters of water) to nasal air flow (12 liters per minute) before and after stimulation.

6. Buhrmester, C. C., and Wenner, W. F.: Presence of a Histamine-like Substance in Nasal Mucosa, Nasal Polypi, and Nasal Secretion, Arch. Otolaryng. **24**:570 (Nov.) 1936.

Not improved with treatment; polypectomy advised.
Not improved with treatment; polypectomy advised.
One month later.

The reactions of the erectile tissue of the inferior turbinate in the nonblocked nose to the experimental agents used were plotted as curves within the normal range of average resistance, estimated between 3.25 and 6.5 mm.

In the obstructed nose all the stimuli with the exception of a 5 per cent hypertonic saline solution induced variable responses plottable beyond the normal limits of resistance, ranging between 6.5 and 400 mm.

A 5 per cent hypertonic saline solution invariably produced a decrease in the degree of vasodilatation, as indicated by a rapid and sustained drop in the resistance curve.

CONCLUSIONS

1. Air pressure of approximately 15 pounds (6.8 Kg.), commonly employed in nasal sprays, produces little or no erectile tissue response in a nonobstructed nose. Air pressure of the same magnitude in an obstructed nose increases both the swelling of the erectile tissue and the degree of nasal obstruction.

2. Tonicities of sodium chloride solution corresponding to strengths of from 0.5 to 5 per cent produce little or no response in a nonobstructed nose. In an obstructed nose isotonic and hypotonic solutions gradually increase the swelling of the erectile tissue, producing a corresponding increase of nasal obstruction. A hypertonic solution of 5 per cent, on the other hand, decreases swelling of the turbinates and nasal obstruction.

INCIDENCE OF ATROPHY OF THE OLFACTORY NERVES IN MAN

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TORONTO, CANADA

The ganglion cells of the olfactory nerves are contained in the epithelium lining the upper part of the nasal cavity, and thus, unlike the ganglion cells of other sensory nerves, they are at the surface of the body and therefore in a comparatively vulnerable position. Situated as they are, they are fairly well protected against trauma but are in considerable danger of being destroyed by inflammatory processes in the nasal mucosa. This has been demonstrated in rats.[1] It has also been shown that these cells do not regenerate[2]; hence, the loss of a certain number of olfactory ganglion cells leads to a corresponding and permanent decrease in the number of olfactory nerve fibers. The question is, how commonly and to what extent are these cells and therefore the olfactory nerve fibers destroyed? The answer to this question is not readily available, on account of the practical difficulties involved; e. g., the whole of the olfactory epithelium is not easily removed for examination, and further, the olfactory nerves do not form a single trunk but are fine filaments which cannot be completely dissected. Therefore, it is necessary to approach the problem by studying the olfactory fibers where they come together on the surface of the olfactory bulb. This was the method adopted in the study to be reported here.

MATERIAL AND METHODS

The olfactory bulbs were obtained from adult human brains which had been removed at autopsies and turned over to the department of anatomy of the University of Toronto as normal specimens. Unfortunately, these brains were not tagged, so the age, sex and other details regarding the subjects from which they were taken are unknown. In a large number, also, the olfactory bulbs were torn or missing. In only 84 of 141 brains were they in good condition, and in 5 of these only 1 bulb was present. Consequently, only 163 specimens were obtained for study. These were embedded in paraffin and sectioned serially at 15 microns. Every twenty-

From the Department of Anatomy, University of Toronto.

1. Smith, C. G.: Pathologic Change in Olfactory Nasal Mucosa of Albino Rats with "Stunted" Olfactory Bulbs, Arch. Otolaryng. **25**:131-143 (Feb.) 1937.

2. Smith, C. G.: Changes in the Olfactory Mucosa and the Olfactory Nerves Following Intranasal Treatment with One Per Cent Zinc Sulphate, Canad. M. A. J. **39**:138-140, 1938.

fifth section was mounted, except in a few cases in which all were mounted in order to insure that the smaller number of sections gave an adequate conception of the condition of the olfactory nerves. Hematoxylin and eosin were used for staining.

OBSERVATIONS

The human olfactory bulb is a superiorly-inferiorly flattened ovoid, approximately 11 by 5 by 2 mm. in size, with its long axis in the sagittal plane. It is rooted to the cribriform plate by the filaments of the olfactory nerves. These nerves reach not only the inferior surface of the bulb but also the whole of its medial and anterior borders and the forepart of its lateral border. In the removal of the brain these filaments are torn off short, and the bulb is left with a granular surface. In certain bulbs, which are much flattened and usually much smaller, this granular area is absent and the whole surface of the bulb is smooth. Stained sections of these flattened smooth bulbs reveal that all olfactory nerves have disappeared (fig. 1, *2*). Thus, there is a possibility of recognizing in the gross, specimens which have lost all their olfactory nerves. For the recognition of the lesser degrees of atrophy, serial sections are required. In these the approximate extent of the loss of the olfactory nerves is in most cases fairly obvious, but in order properly to classify the bulbs some means of measuring this atrophy had to be found. A consideration of the structure of the olfactory bulb will explain the basis of the method adopted.

Figure 1, *1* is a cross section through the anterior third of a normal bulb. On the surface can be seen the layer of fibers formed by the terminating olfactory nerves. From the deep aspect of this layer bundles of fibers pass inward to end in closely woven masses, called olfactory glomeruli. Within these glomeruli the olfactory fibers synapse with the dendrites of the mitral cells, whose cell bodies, as seen in a cross section, are posted in single file at the periphery of the mass of more or less closely crowded small cells called the internal granular layer. The cells of the corresponding external granular layer are few and are to be found in the region of the glomeruli. Since the glomeruli represent the endings of the olfactory nerve fibers, their number decreases in proportion to the decrease in the nerve fibers; i. e., contrary to the finding in rats,[3] a glomerulus disappears as soon as all olfactory fibers entering it degenerate, as is evident in figure 1 (*2* and *3*). In *2* all the olfactory nerves have degenerated and all the glomeruli have also disappeared. In *3* most of the olfactory nerves have degenerated and a correspondingly large fraction of the glomeruli have been lost; those that

3. Smith, C. G.: The Changes in Volume of the Olfactory and Accessory Olfactory Bulbs of the Albino Rat During Postnatal Life, J. Comp. Neurol. **61**: 477-508, 1935.

remain are found only at the termination of the sole remaining bundle of olfactory nerve fibers. Thus, a count of the glomeruli may be expected to yield a value which can be used as an index of the number of olfactory fibers reaching the bulb. It should be pointed out that, although the

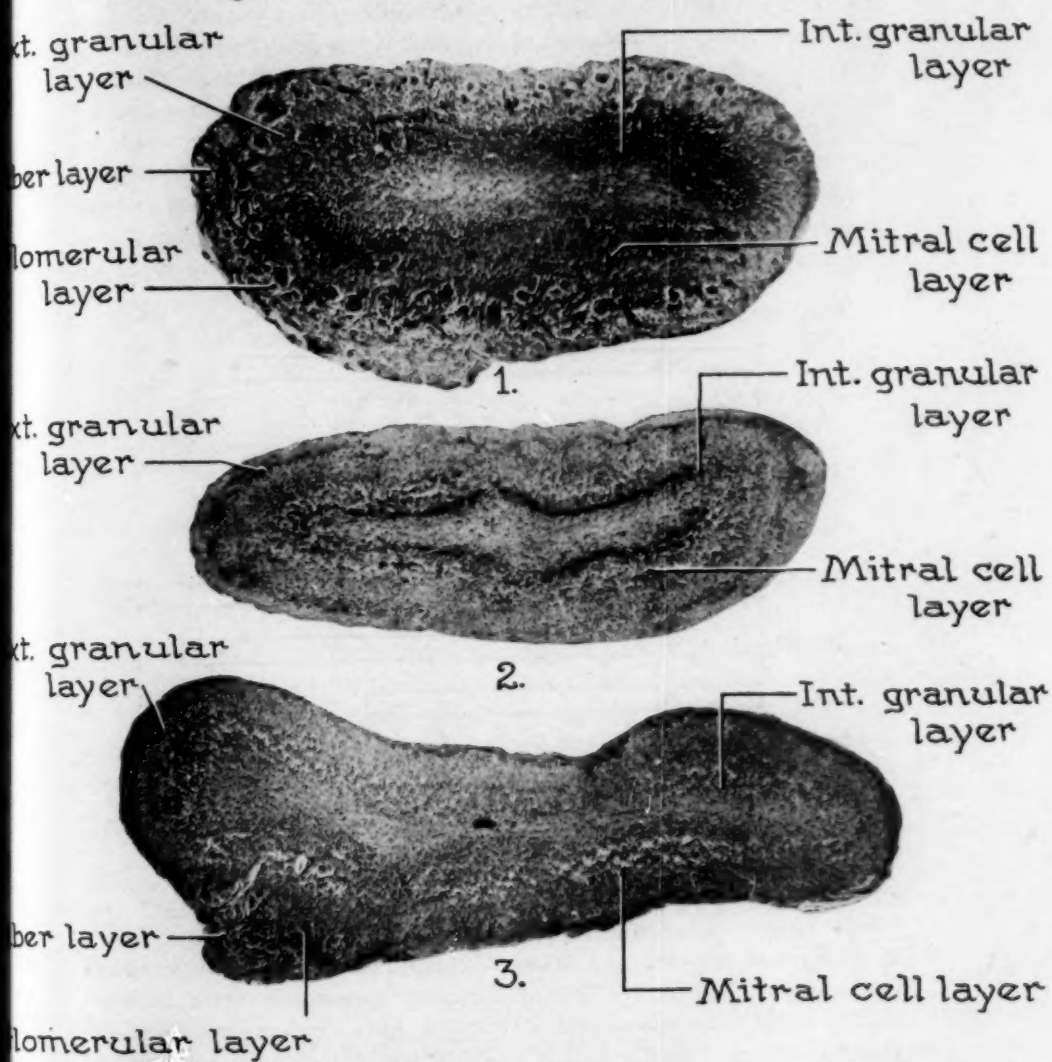

Fig. 1.—*1*, cross section through the anterior third of a normal olfactory bulb (× 21). *2*, cross section through the anterior third of a completely atrophic bulb (× 21). *3*, cross section through the middle of a bulb in which 95 per cent of the olfactory nerves have atrophied (× 21).

glomeruli vary in size and therefore do not always receive the same number of olfactory fibers, the range in size is similar in both atrophic and

normal bulbs; hence, the average number of fibers per glomerulus (n) is approximately the same for all bulbs.

The total number of glomeruli was determined by counting those in the mounted sections and multiplying the result by twenty-five, since only every twenty-fifth section was saved. On studying the total numbers of glomeruli obtained in this way, it was found that the normal bulbs (i. e., those which from inspection of the sections were estimated to have a full complement of olfactory nerves and which possessed healthy-looking glomeruli) had a count of 10,000 or more, while all atrophic bulbs had a lesser value, depending in each case on the number of olfactory fibers still present. Since 10,000 was the smallest number of glomeruli found in a normal bulb, this number multiplied by n, (the average number of fibers per glomerulus) was taken to represent

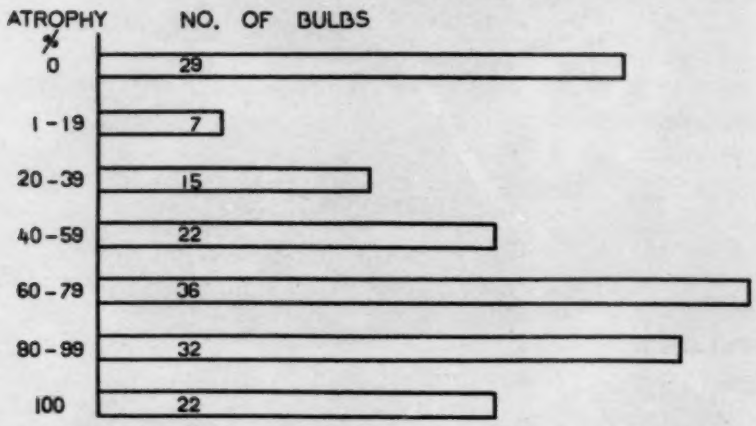

Fig. 2.—Chart showing the incidence of atrophy of the olfactory nerves in 163 olfactory bulbs.

a full complement of olfactory fibers. Any bulb with less than 10,000n olfactory fibers was rated as atrophic. That is, a bulb with 9,000 glomeruli and hence 9,000 n olfactory fibers was rated as 10 per cent atrophic; one with 8,000 glomeruli and hence 8,000 n olfactory fibers, as 20 per cent atrophic, and so on. Actually, since partially degenerated glomeruli, identifiable by their moth-eaten appearance, were included in the count, the amount of atrophy is somewhat greater than the results indicate. Figure 2 is a chart in which the bulbs are grouped according to the degree of atrophy, e. g., 0 per cent, 1 to 19 per cent, 20 to 39 per cent, and the incidence in each group is plotted. It is seen that among the 163 bulbs there were only 29 normal specimens, while in 22 all the olfactory nerves had degenerated. These completely atrophic bulbs were obtained from 16 brains. In 6 of these the condition was bilateral and in 8 unilateral, while in 2 brains only 1 bulb was

available, and it was atrophic. The chart also shows that of the 112 bulbs in which the loss of olfactory nerves was only partial, 7, 15, 22, 36 and 32 had lost, respectively, 1 to 19 per cent, 20 to 39 per cent, 40 to 59 per cent, 60 to 79 per cent and 80 to 99 per cent of their olfactory nerves. These data reveal that 90, or 55 per cent, of the 163 bulbs had lost more than three fifths of their complement of olfactory nerves. Since normally both sides of the nose are used in detecting an odor, the loss of olfactory nerves per brain rather than per bulb is also of interest. The results for 79 brains in which both bulbs were present are as follows: Eleven were normal; 7 had lost 1 to 19 per cent; 5, 20 to 39 per cent; 13, 40 to 59 per cent; 15, 60 to 79 per cent; 22, 80 to 99 per cent, and 6 had lost all their olfactory nerves. That is, 43 of 79 brains, or 53 per cent, had suffered a loss of more than three fifths of their olfactory nerves.

These findings make it of interest to determine whether there is a tendency for atrophy to occur in a certain part of the bulb. To this end the location of the atrophy was recorded in all cases. The observations reveal that in the greater number, i. e., 66, the atrophy was not localized. In 31 the atrophy was maximal at the anterior end and in 8 at the posterior end, and in 7 both the anterior and the posterior ends were atrophic. It would appear, then, that if the atrophy is localized, it is more frequently found at the anterior end and that the last fibers to survive are those that pass to the middle of the bulb, as in figure 1, *3*.

A comparison of the incidence of atrophy in right and left bulbs showed that there was no tendency for one side to be involved more than the other. Thus, the average amount of atrophy in 82 right bulbs was 55 per cent and in 81 left bulbs 58 per cent. A comparison of the atrophy present in the two bulbs removed from a single brain showed that the atrophy tended to be equal on the two sides, although a considerable difference could occur. Thus, in 53 brains the amount of atrophy in the two bulbs did not differ by more than 19 per cent, in 19 the difference was 20 to 39 per cent, in 6 the difference was 40 to 59 per cent and in 1 the difference was 74 per cent.

COMMENT

The results of this study indicate that it is not unusual for a person who has reached maturity to have lost a big part of his quota of olfactory nerves. This finding is rather unexpected in view of recent work by Elsberg and his associates,[4] in which the sense of smell was found,

4. Elsberg, C. A.; Levy, I., and Brewer, E. D.: A New Principle for the Classification of Odors Based upon Their Olfactory Coefficients, Bull. Neurol. Inst. New York **4**:20-25, 1935.

with few exceptions, to be unimpaired and about the same in all normal adults. Anatomic studies, however, though few, are in agreement with this study in that they tend to show that a normal olfactory sense organ is relatively uncommon. Suchannek [5] pointed out that sensory olfactory cells may be destroyed by inflammatory processes which spread into the olfactory mucosa, and that once destroyed they are replaced by nonsensory ciliated columnar cells. Brunn [6] cited studies by Welcker, Henle and Waldeyer in which no sensory olfactory epithelium was found on examination of the well preserved mucosa of 4 men. Brunn studied the nasal mucosa of 4 executed persons who had been in apparent good health; in 1 specimen he found no olfactory cells, while in 2, in which the extent of the sensory epithelium was carefully mapped, he found this to be small and invaded by numerous streamers of ciliated columnar epithelium. Kolmer [7] also noted this condition in many of his cases and concluded that the olfactory area is commonly small in adult man because it has been partially destroyed by the inflammatory processes which may repeatedly invade it. It is of interest that, although Kolmer felt that in many of his cases the olfactory area was thus secondarily reduced in size, he had no way of telling what the original extent of the olfactory area had been. In my study of the olfactory nerves this difficulty did not occur. That is, it was always possible to recognize the area of the bulb which once had received olfactory nerves even if all the nerves had atrophied. This was possible because the mitral cells, which synapse with the olfactory nerve fibers and help to form the glomeruli, always remain. Thus, whereas Kolmer could not estimate the amount of atrophy of the olfactory area and consequently of the olfactory nerves, my approach to the problem makes this possible. The results bear out his contention that the olfactory area in adults is often much smaller than it was in early childhood.

The question arises as to whether the incidence of atrophy of the olfactory nerves found in the studies reported here may not be too high, owing to an unintentional selection in collecting the material. That is to say, atrophic bulbs, having lost the nerve fibers connecting them with the nasal mucosa, may be less adherent to the dura than normal bulbs and hence more readily removed intact. It is known that the 163 bulbs used in this study were obtained from 141 brains; hence, allowance can

5. Suchannek, H.: Differentialdiagnostische Merkmale zur Unterscheidung zwischen normalen und pathologischen menschlichen Riechepithel, resp. respiratorischem Flimmerepithel, Ztschr. f. Ohrenh. **22**:4-10, 1891.

6. Brunn, A.: Beiträge zur mikroskopischen Anatomie der menschlichen Nasenhöhle, Arch. f. mikr. Anat. **39**:632-651, 1892.

7. Kolmer, W.: Geruchsorgan, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 3, pp. 192-249.

be made for the possible tendency to discard normal bulbs by calculating the incidence of the different degrees of atrophy on the basis of a total of 282 bulbs. Thus, assuming that the majority, two thirds, of the unstudied bulbs were normal, the normal bulbs, i. e., 109, would still make up only 39 per cent of the total. Further, assuming that none of the unstudied bulbs had lost as much as one half of their olfactory nerves, the fraction of the total number of bulbs with a 50 per cent reduction in the number of olfactory nerves would still be 35 per cent.

One cannot help wondering if disappearance of these nerves in adults does not help to close the portal of entry for neurotropic viruses, even as cutting of the olfactory tracts in monkeys [8] protected against nasal instillation of poliomyelitis virus.

SUMMARY

Serial sections of 163 olfactory bulbs were prepared with a view to studying the incidence of atrophy of the olfactory nerves. The observations reveal that atrophy of these nerves is common in adults—55 per cent of these bulbs had lost more than three fifths of their complement of olfactory nerves and 13 per cent had lost all their nerves. Only 29 bulbs were considered to have a normal complement of olfactory nerve fibers. The number of olfactory nerve fibers reaching a bulb was estimated from the total number of glomeruli. This was possible because the average number of fibers terminating in a glomerulus is believed to be constant and the glomerulus disappears when all the olfactory fibers which terminate in it have degenerated. For this study a normal bulb was one which was estimated to contain 10,000 or more glomeruli. Partial atrophy of olfactory nerves may occur in all parts of a bulb or be localized to the anterior or, less commonly, to the posterior end. The last fibers to survive are those that pass to the middle of the bulb. There was no tendency for atrophy to occur more frequently on one side of the nose than on the other. The two bulbs for a given brain usually showed a similar degree of atrophy, but in certain cases the difference on the two sides was considerable. The atrophy of olfactory nerves is believed to be due to destruction of the olfactory ganglion cells located in the nasal mucosa by the inflammatory processes that occur in the mucosa. Recent studies of the sense of smell do not reveal the impairment which the high incidence of atrophy of the olfactory nerves might lead one to expect.

8. Schultz, E. W., and Gebhardt, L. P.: Olfactory Tract and Poliomyelitis, Proc. Soc. Exper. Biol. & Med. **31**:728-730, 1934.

USE OF PROSTIGMINE FOR IMPAIRED HEARING

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NEW YORK

In 1939 Davis and Rommel[1] reported benefit from the use of prostigmine in the treatment of acute and chronic deafness and also in relieving tinnitus aurium. The present study of 30 patients with impaired hearing was made to determine what improvement, if any, can be obtained by the use of this drug.

Twenty-four of the patients studied had had loss of hearing over a long period of years. Five patients had had impaired hearing for periods not longer than six months. Two of these had a very recent involvement. Twenty-three had associated tinnitus.

These patients were from the outpatient department of Gouverneur Hospital and from private practice.

Before treatments were started, each patient was given an examination of the nose, throat and ears and an audiometric reading was made with a Western Electric 6-A audiometer. The tests were made in rooms adjoining the clinic or office. There was an ordinary amount of noise from adjacent activities, although an attempt was made to reduce the noise as much as possible. A sound-proof room was not used. No attempt was made to perform the audiometric tests under any particular conditions of weather or temperature. To avoid fatigue, the tests were made as soon as the patient arrived. During the tests there were long pauses.

The audiometer was calibrated before each test. The signals were introduced from 0 downward, and the decibel reading when the signal was first heard was noted. This reading was rechecked several times but at irregular and arrhythmic intervals. No reading was noted that had been obtained by the introduction of a very loud tone, gradual diminution of the tone and determination of the decibel reading at which the tone faded entirely.

The treatments began with the application of a large nasal tampon saturated with a 10 per cent solution of mild protein silver. This was left in place for thirty minutes, and its removal was followed by irrigation and suction (Dowling). Then an intramuscular injection of 1 cc. of a 1:2,000 solution of prostigmine methylsulfate was administered.

1. Davis, T. C., and Rommel, J. C.: Treatment of Deafness and Contiguous Nervous Disorders with Prostigmine: Preliminary Clinical Report, Arch. Otolaryng. **29**:751-758 (May) 1939.

For children I used a 1:4,000 solution. The treatments were given three times a week or as close to that as could be managed.

Since most of the conditions treated were of long standing and all the patients, according to their histories, had been subjected to eustachian catheterization, massage of the openings in the pharynx and other therapeutic procedures, such treatments were not given by me.

Hughson and Witting [2] said: "However many arguments may be advanced that threshold audibility for discrete tonal frequencies is not a true estimate of speech hearing, nevertheless these threshold determinations do indicate a change of hearing at these low levels and as such can be used to measure the effect of therapy."

Ordinarily, a change of 5 decibels would be accepted as a margin for error. For patients in whose cases the decibel reading was checked and rechecked a number of times and at irregular intervals, it is permissible to consider a variation of 5 decibels as at least a definite change. However, patients in whose cases changes were limited to 5 decibels or less were not included among those considered "improved." The frequencies of 512, 1024, 2048 and 4096 were the only ones used in computing the changes.

It should be noted that in those cases in which the decibel reading shows a small improvement, any subjective improvement which is apparent to the patient is of value from a psychologic angle. Often such a patient has been examined and treated for various periods in various manners and has then been dismissed with a hopeless prognosis. Thus, any subjective lessening of the impairment of hearing has a constructive value. Some patients are so eager to get better that they will overcooperate with the examiner and so by their very eagerness negate the result of the audiometer tests and produce false readings. This can be eliminated by repeated checks of audiometer readings at irregular intervals.

In the cases studied I also noted the effect of prostigmine on other parts of the body. In no case was there cardiac involvement as expressed by shortness of breath, dyspnea, dizziness or tachycardia. Pritchard [3] noted that atropine was given to counteract the effect of prostigmine on the bowel in myasthenia gravis. In none of my cases were any ill effects on the bowel noted. Two patients complained of a slight looseness of the bowels for a few days. This condition did not recur in any case for the duration of the treatment.

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2. Hughson, W., and Witting, E. G.: Estimation of Improvement in Hearing Following Therapy of Deafness, Ann. Otol., Rhin. & Laryng. **46**:368-383 (June) 1940.

3. Pritchard, E. A. B.: Use of "Prostigmin" in Treatment of Myasthenia Gravis, Lancet **1**:432-434 (Feb. 23) 1935.

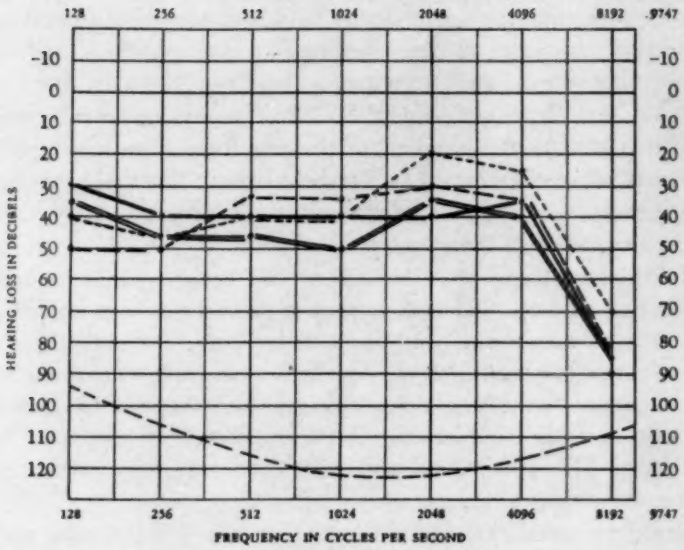

Chart 1 (case 1).—W. E., a white woman aged 40, a school teacher, gave a history of loss of hearing on both sides for the past four years and constant buzzing in both ears for the past twenty-five years. The loss of hearing had become progressively worse. She wore a hearing aid (radio tubes). Twenty years ago she underwent a tonsil operation. She had had various treatments of the ears, including catheterization, without any benefit. The family history was not significant. Examination showed the nose to be essentially normal. There was a postnasal discharge. Both drum membranes were cloudy over their entire extent, retracted and movable. Nasal tampons saturated with a 10 per cent solution of mild protein silver were applied and a solution of prostigmine methylsulfate (1:2,000) was injected three times a week until fifty-three treatments had been given. Results noted subjectively were marked diminution of the buzzing, beginning after the second month of treatment, and improvement of the hearing when a hearing aid was not used. Results determined objectively were: right ear—initial loss 39 decibels, terminal loss 36, gain 3; left ear—initial loss 42.5, terminal loss 31, gain 10.5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Jan. 6, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on July 15.

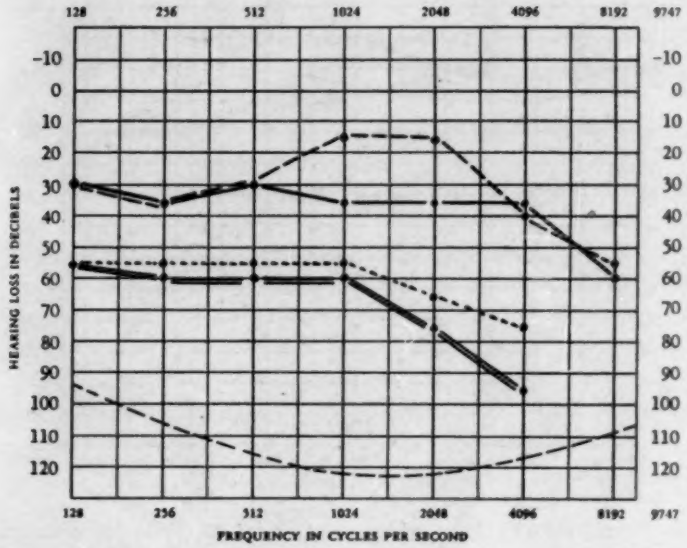

Chart 2 (case 2).—G. E., a white woman aged 23, had had constant buzzing in both ears, with marked impairment of hearing on both sides, more marked on the left, for the past seven years. During childhood she had frequent colds in the nose and stuffiness of both ears, which cleared up. Her history revealed that a variety of treatments of the ears produced no change and that her hearing had become worse. The family history was noncontributory. On examination the patient had some nasal twang to her voice and spoke in a flat monotone. The middle turbinates on the right were hypertrophied. A discharge was present in the middle meatus on both sides. The tonsils were small and submerged. Both drum membranes were thick, cloudy, retracted and movable. Nasal tampons saturated with 10 per cent solution of mild protein silver were applied and a solution of prostigmine methylsulfate (1:2,000) was injected three times a week until thirty-six injections had been given. At the time of writing the patient says she hears better than she did before the treatments and can carry on ordinary conversations without difficulty. The buzzing has diminished markedly. Results determined objectively were: right ear—initial loss 34 decibels, terminal loss 25, gain 9; left ear—initial loss 72.5, terminal loss 62.5, gain 10.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Jan. 20, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on March 7.

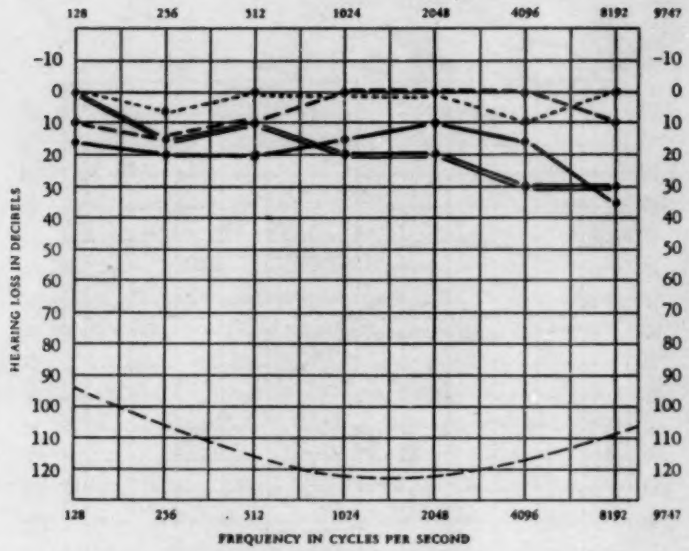

Chart 3 (case 3).—G. L., a white woman aged 26, came for examination on Jan. 14, 1940 with the complaint of having had a cold for the past three weeks, accompanied with sore throat and pain in the right ear for the past three days. She said that the pain in the ear had been getting worse. There were large crusts in the nose, with a thick discharge. She had to clear her throat frequently and had a constant buzzing in the right ear. There was a history of frequent colds in the nose and frequent sore throat. Ten years before, she had had a tonsil operation. There was no family history of deafness. Examination of the nose revealed hypertrophy of the middle turbinates, with a bilateral mucoid discharge. There was a thick postnasal discharge. The ear drums appeared to be normal. For one month a 10 per cent solution of mild protein silver was applied on nasal tampons. The condition cleared up, except the buzzing in the ears, which continued. Hearing tests revealed the loss shown on the chart. Beginning on February 13, the patient received applications of mild protein silver on nasal tampons and injections of a solution of prostigmine methylsulfate three times a week until thirty-six injections had been given. The buzzing in the right ear cleared up entirely and at the time of writing the patient says she hears well—"better than ever." Results determined objectively were: right ear—initial loss 22 decibels, terminal loss 12.5, gain 9.5; left ear—initial loss 22.5, terminal loss 17.5, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on February 13. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on May 13.

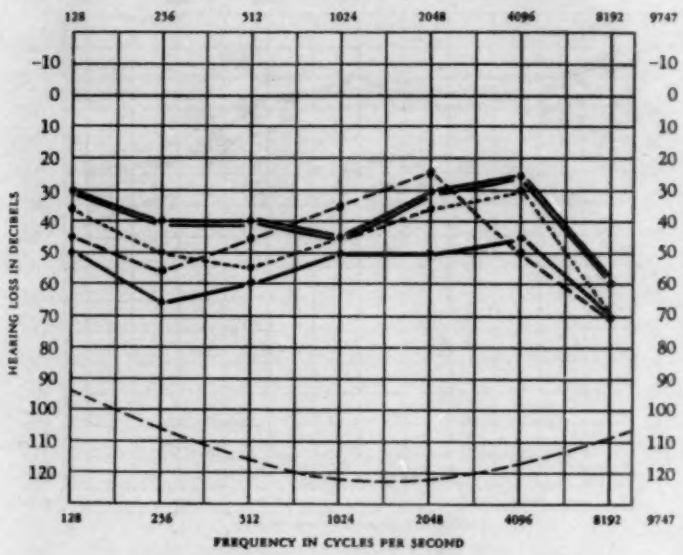

Chart 4 (case 4).—M. B., a white woman aged 37, came for examination on Dec. 14, 1938 with the complaint of frontal headaches of a number of years' duration. These had been treated, with no improvement. She had frequent colds in the nose, which became stuffed. Her throat was dry. She had had defective hearing as long as she could remember. A long time ago she had a tonsil operation. She had had treatment for headaches and impaired hearing. The family history revealed that her grandmother and an aunt on the paternal side were deaf. Examination showed the middle turbinates on both sides to be hypertrophied. There was a bilateral nasal discharge. The drum membranes of both ears were gray and retracted. Nasal tampons saturated with a solution of mild protein silver were applied three times a week for two months. Clinical improvement of the condition of the nose and abatement of the headaches were obtained. On Dec. 10, 1939 the patient returned for treatment to the ears and was given seven injections of a solution of prostigmine methylsulfate, and nasal tampons were applied. She stopped coming for treatments, and I have been unable to get in touch with her since. Results determined objectively were: right ear—initial loss 51 decibels, terminal loss 39, gain 12; left ear—initial loss 35, terminal loss 41, gain 6.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 10, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on December 22.

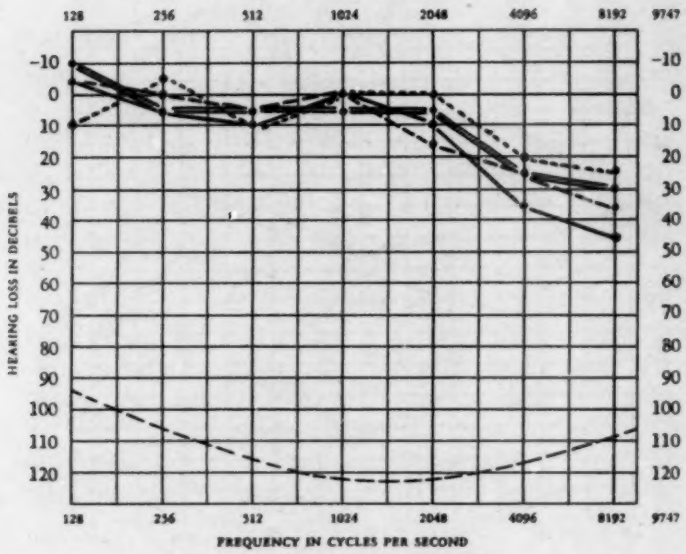

Chart 5 (case 5).—Mrs. M. H., a white woman aged 38, wanted to reduce. She took thyroid for three months and adhered to a sugar-free and starch-free diet. She began to have dizzy spells, with nausea and vomiting, and on one occasion fainted. She had a constant sensation of pounding in the head, with impaired hearing and buzzing. Her past history and the family history were noncontributory. Examination of the nose revealed bilateral hypertrophy of the turbinates. The tonsils were diseased and submerged. The ears were normal. The Kobřak test, with 5 cc. of water of 45 F., produced a hyperactive reaction on both sides.

A 10 per cent solution of mild protein silver was applied on nasal tampons and injections of a solution of prostigmine methylsulfate were given three times a week until thirty-six treatments had been given. After thirteen treatments the patient made the comment that the tinnitus had decreased markedly, that she experienced no nausea, vomiting or fainting and the dizziness was only occasional and not marked. The dizziness and tinnitus cleared up entirely. Her family noticed that her hearing was better. Results determined objectively were: right ear—initial loss 14 decibels, terminal loss 5, gain 9; left ear—initial loss 10, terminal loss 4, gain 6.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 28, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on March 12, 1940.

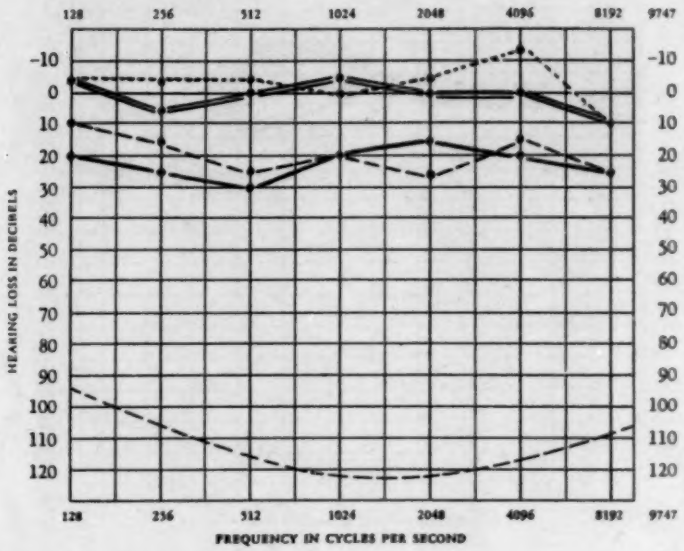

Chart 6 (case 6).—Miss R. J., a white woman aged 35, came for examination with a complaint of loss of hearing in both ears. After wax had been removed from both ears, there was a continued loss of hearing on the right side only. There was a history of frequent colds in the nose, and the patient had an irritating tickling in the throat. She had had nosebleeds which required surgical treatment. There had been frequent blocking of both ears with wax. When this was removed, the hearing was improved. The patient thought the hearing on the right had never been as good as that on the left. There was no family history of deafness. Examination revealed a discharge in both sides of the nose and a postnasal discharge. Both drum membranes were slightly cloudy. A 10 per cent solution of mild protein silver was applied on nasal tampons and an injection of a solution of prostigmine methylsulfate was given three times a week until thirty-six treatments had been given. At the time of writing, the patient is not aware of any loss of hearing on either side and is conscious of improved hearing on the right. Results determined objectively were: right ear—initial loss 21 decibels, terminal loss 21, gain 0; left ear—initial loss + 1, terminal loss + 6, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 11, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on April 30, 1940.

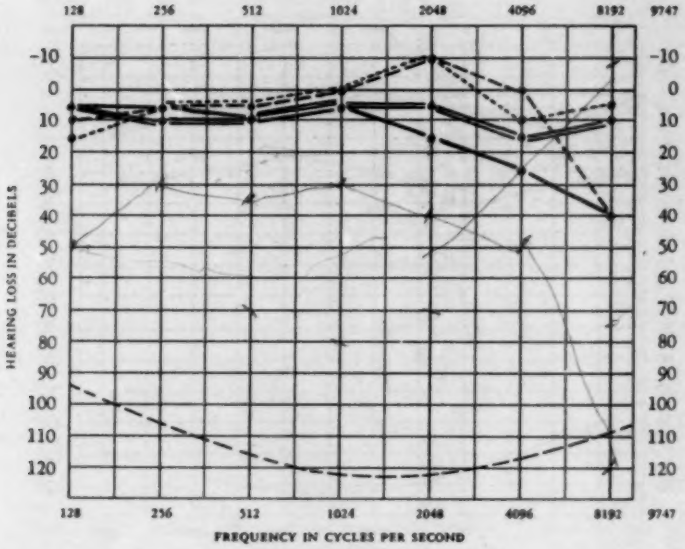

Chart 7 (case 7).—F. V., a white man aged 30, came for treatment on June 6, 1939, complaining of headaches, frequent colds and postnasal discharge. The past history revealed that he had had congenital syphilis, for which he received treatment until the Wassermann reaction was negative. There was no family history of hearing loss. His mother was psychotic and syphilitic. On examination there was a discharge in the nose and in the postnasal region. The ears were normal. On November 24 the patient noted loss of hearing on the right, which, although not marked, was sufficiently noticeable that he had to ask people who spoke to him to repeat. This difficulty had been present for a long time.

From June 6, 1939 until the date of discharge the patient was treated with a 10 per cent solution of mild protein silver applied on nasal tampons. Beginning on November 24, he was given injections of a solution of prostigmine methylsulfate at the rate of three a week until he had received thirty-six injections. After these treatments he said that his hearing was good and had improved so that it was not necessary any more to ask people to repeat. Results determined objectively were: right ear—initial loss 14 decibels, terminal loss 1, gain 13; left ear—initial loss 9, terminal loss 1, gain 8.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 24, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on May 7, 1940.

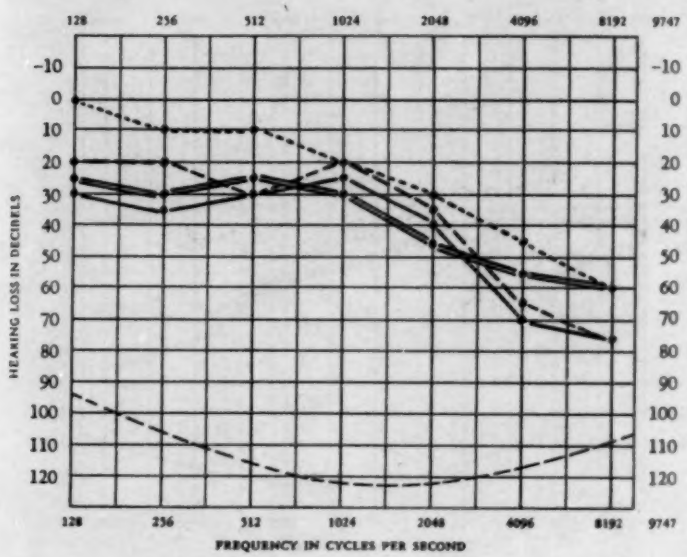

Chart 8 (case 8).—A. H., a white man aged 67, gave a history of aural involvement for the past fifty years. At various intervals he had had several attacks of purulent otitis media, the last about ten years previously. This lasted about three weeks. For the past three years he had been treated for tinnitus, which was constant, and for loss of hearing on the left, which became progressively worse. Treatment was given to the nose and by catheterization. The family history revealed that his father was hard of hearing. Examination showed the nose and throat to be essentially normal. The throat showed some postnasal discharge. Both drum membranes were thick and cloudy and showed scattered calcareous deposits. The patient was given thirty-six injections of a 1:2,000 solution of prostigmine methylsulfate at the rate of three a week. He reported that the right ear felt less clogged and that the tinnitus abated periodically. At the time of writing he can hear in the moving picture theaters to a considerable extent but generally finds his hearing impaired, as before the treatments. Results determined objectively were: right ear—initial loss 41 decibels, terminal loss 37.5, gain 3.5; left ear—initial loss 39, terminal loss 26, gain 13.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 13, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on April 27, 1940.

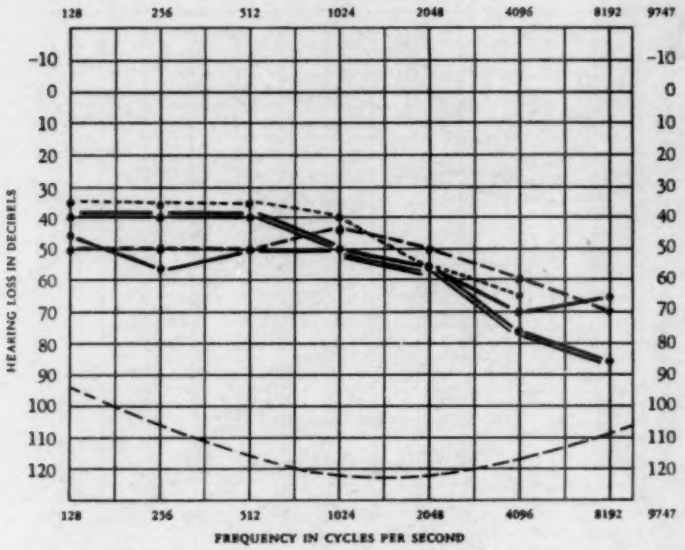

Chart 9 (case 9).—R. A., a white man aged 53, gave a history of progressive loss of hearing on both sides, associated with constant tinnitus, for the past twenty-five years. During the past winter he had had attacks of vertigo, associated with marked buzzing, which cleared up under treatment (nasal tampons saturated with a solution of mild protein silver). At various times he had a profuse discharge from the nose and he had to clear his throat frequently. The past history revealed that he had had a tonsillectomy about twenty-eight years previously and that at various times he has been treated locally for chronic sinusitis. There was no family history of impaired hearing. Examination of the nose showed a mucopurulent discharge on both sides. There was a postnasal drip. Both drum membranes were thick, cloudy and retracted. After about twenty-four treatments the patient said that the buzzing was almost gone, but he later reported that it returned at intervals and had returned to its former severity but was not constant. At the time of writing he does not notice any improvement in his hearing. Results noted objectively were: right ear—initial loss 54 decibels, terminal 51, gain 3; left ear—initial loss 55, terminal 49, gain 6.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 3, 1939. The dash line indicates the reading for the right ear and the dotted line that for the left ear, on March 9, 1940.

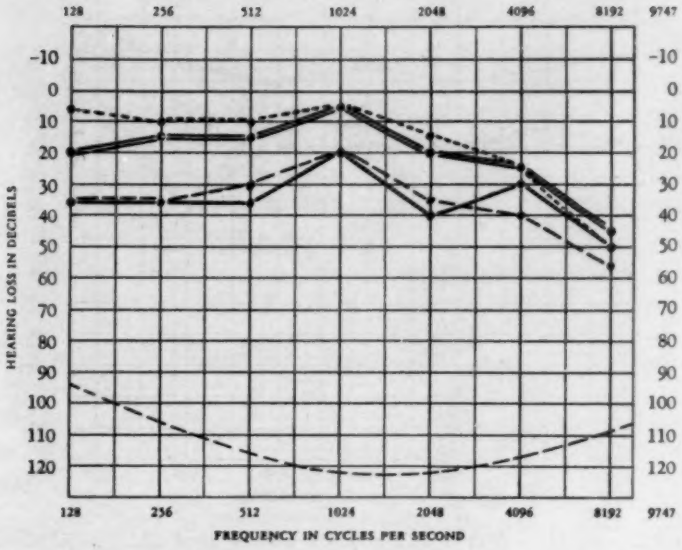

Chart 10 (case 10).—G. D., a white man aged 54, had been under treatment since 1927 for atrophic rhinitis. He had large crusts in the nose. His sense of smell was impaired, and he had headaches, for which he had been treated. He had noted gradual loss of hearing on the right. Examination showed the drum membranes to be thin, retracted, somewhat cloudy and movable. The tonsils were small and submerged. The nasal mucosa was atrophic. He was given seventy-one treatments, consisting of the application of nasal tampons saturated with a solution of mild protein silver and the injection of a solution of prostigmine methylsulfate. At the time of writing, the patient says he hears better on the right and is not conscious of any difference of hearing on the two sides. Results determined objectively were: right ear—initial loss 31 decibels, terminal loss 31, gain 0; left ear—initial loss 16, terminal loss 14, gain 2.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, before treatment. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, after treatment.

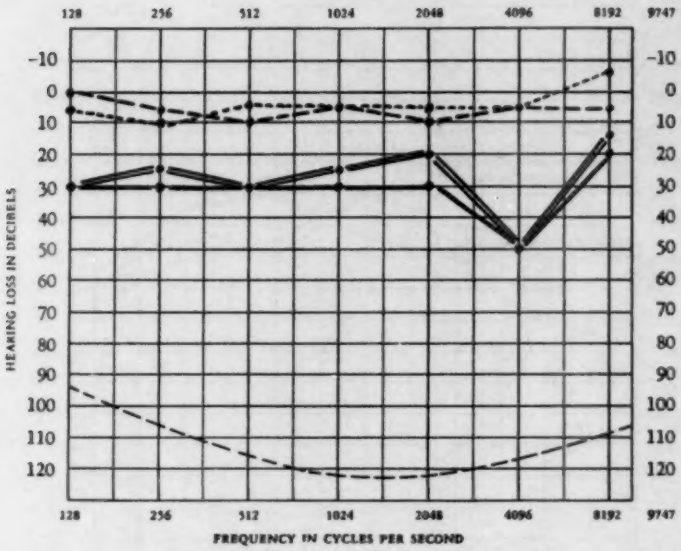

Chart 11 (case 11).—Mrs. P. C., a white woman aged 27, complained of progressive loss of hearing in both ears, associated with constant roaring and buzzing, for the last five months. About six months previously she had a "cold," which lasted for three weeks. Several weeks later, the buzzing and loss of hearing occurred. There was no complaint referable to the nose and throat except occasional postnasal discharge. The past history was noncontributory. Examination revealed mucoid discharge in both sides of the nose. The tonsils were diseased and hypertrophied. There was a mucoid postnasal discharge. Both drum membranes were slightly cloudy, retracted and movable. Fifteen treatments, consisting of the application of nasal tampons saturated with a solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate were given. At the time of writing, the buzzing has completely disappeared. The patient hears better than at any previous time. Results determined objectively were: right ear—initial loss 32.5 decibels, terminal loss 7.5, gain 25; left ear—initial loss 29, terminal loss 5, gain 24.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, before treatment. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, after treatment.

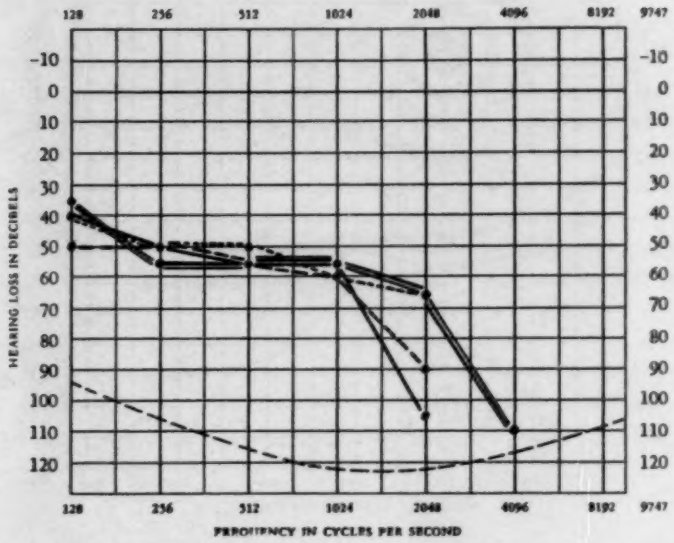

Chart 12 (case 12).—B. R., a white man aged 48, gave a history of bilateral loss of hearing for the past eight years. The onset was gradual. The loss had become most noticeable in the past three years. There was no tinnitus at any time. Examination revealed that both drum membranes were slightly retracted. The septum was deviated to the right, occluding the lumen. The tonsils were diseased and submerged. There was a thick mucoid postnasal drip. The past history showed that the patient had received all kinds of treatments, including catheterization, over a long period. Twenty-four treatments were given at the rate of three a week. These consisted of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. At the time of writing the patient reports that he "still has great difficulty in hearing." Results determined objectively were: right ear—initial loss 84 decibels, terminal loss 81, gain 3; left ear—initial loss 71, terminal loss 74, loss 3.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on March 12, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on May 13.

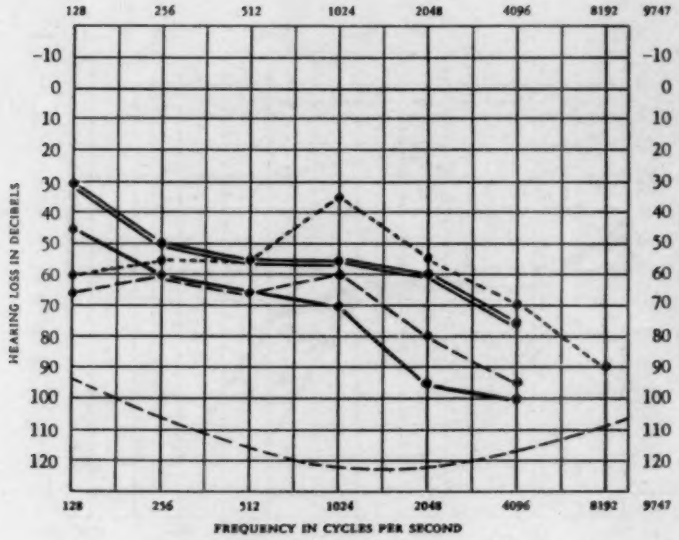

Chart 13 (case 13).—H. E., a white man aged 32, had noticed impairment of hearing on both sides for the past four years. He had received various treatments. At the time he was examined he wore a hearing aid. He reported occasional buzzing and frequent headaches. The family history revealed that his mother and sister had marked impairment of hearing. Examination of the nose showed bilateral hypertrophy of the middle turbinates and mucoid discharge in both middle meatuses. The drum membranes of both ears were cloudy over their entire extent, with scattered calcareous deposits. They were markedly retracted and were movable. There was a mucoid postnasal discharge. Forty-four treatments, given at the rate of about three a week, consisted of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. At the time of writing, the patient has noticed a slight improvement in hearing but still has to use a hearing aid on the left side. Results determined objectively were: right ear—initial loss 82.5 decibels, terminal loss 75, gain 6.5; left ear—initial loss 61, terminal loss 54, gain 7.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Feb. 21, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on June 15.

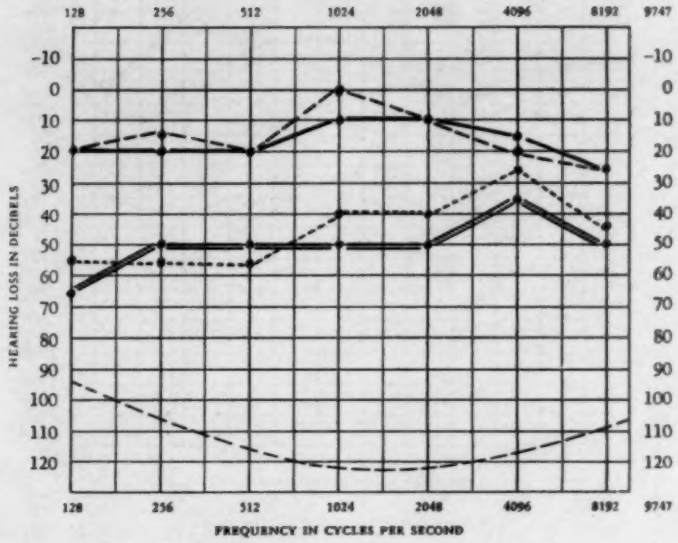

Chart 14 (case 14).—F. R., a white woman aged 50, came for treatment two years ago with a complaint of gradually increased loss of hearing on the left. Examination revealed pus in both middle meatuses. The tonsils were small, submerged and diseased. There was a postnasal discharge. Both drum membranes were cloudy, retracted and movable. Commencing on Nov. 18, 1939, the patient received 23 treatments at the rate of three a week. These consisted of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. At the time of writing, the patient reports that she can hear better at times. Results determined objectively were: right ear—initial loss 14 decibels, terminal loss 12.5, gain 1.5; left ear—initial loss 61, terminal loss 54, gain 7.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on November 18. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on December 23.

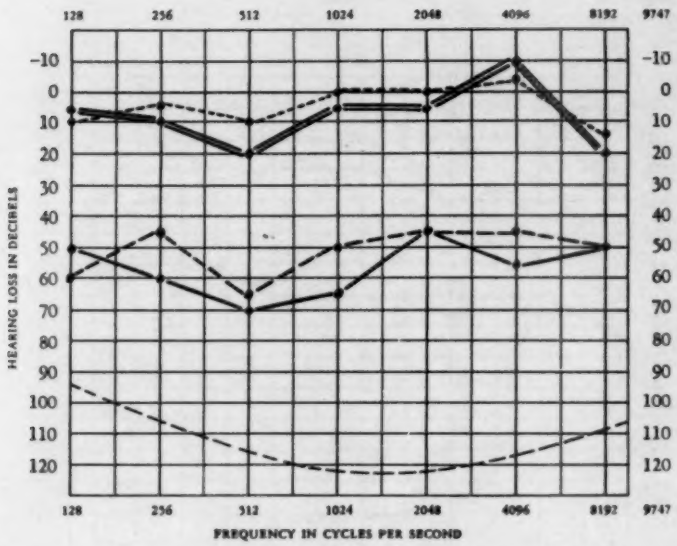

Chart 15 (case 15).—Dr. A. P., a white man aged 40, noted impairment of hearing on the right one year before examination. When he came for examination, he had total loss of hearing on the right but no tinnitus. He had continuous colds in the nose, with constant postnasal discharge. The family history revealed that the patient's father, 2 brothers, 2 sisters and a paternal aunt showed marked impairment of hearing. Examination showed the septum deviated to the right. There was a mucopurulent nasal discharge. The tonsils were diseased and hypertrophied, and there was a mucoid postnasal discharge. At irregular intervals from Nov. 20 to Dec. 18, 1939 the patient received 8 treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. He reported "some improvement on the right." Results determined objectively were: right ear—initial loss 59 decibels, terminal loss 51, gain 8; left ear—initial loss 5 decibels, terminal loss 1, gain 4.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 17, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on Jan. 16, 1940.

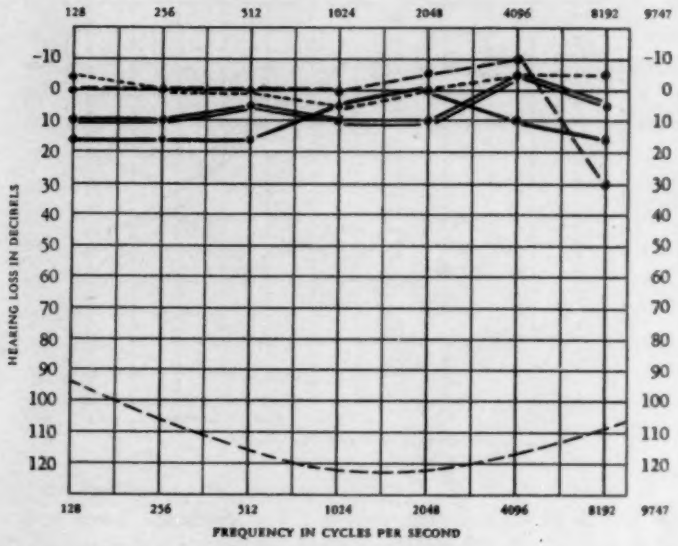

Chart 16 (case 16).—Mrs. P., a white housewife aged 35, complained of buzzing in both ears for the past three months, following a cold in the head. No impairment of hearing was noticeable. Her past history was essentially normal. The results of the examination of the nose and throat were negative. Both drum membranes were somewhat retracted, cloudy and movable. Twenty-seven treatments were given, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. At the time of writing, the patient reports that the buzzing has completely disappeared and that her hearing on both sides is better than before. Results determined objectively were: right ear—initial loss 7.5 decibels, terminal loss +4, gain 11.5; left ear—initial loss 5, terminal loss 0, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on May 25, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on October 4.

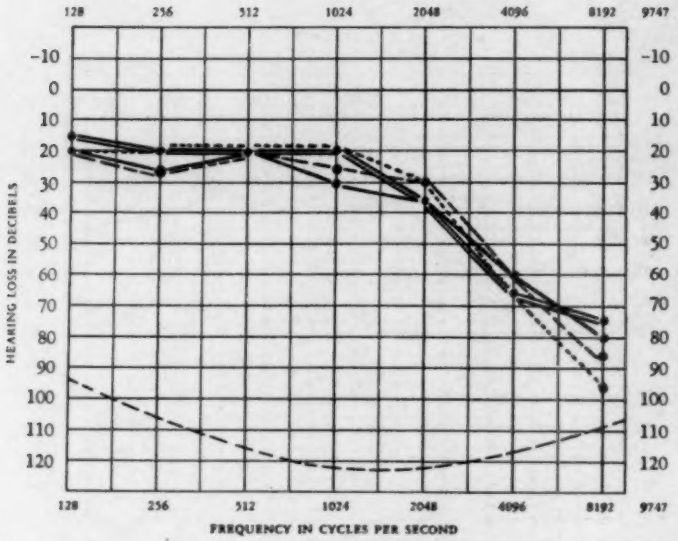

Chart 17 (case 17).—M. J., a white man aged 59, had a discharge from the right ear three weeks before examination. The discharge lasted a few days. After that he experienced a buzzing sound, itching and some impairment of hearing in the right ear. He had no complaint of the left ear. He had a history of cold in the nose several times a year, and at times he had a thick scanty nasal discharge. His past history revealed that in childhood his right ear was accidentally punctured and that at long intervals since then he had had an aural discharge, lasting a short time, for which he had received treatment on various occasions. He had had a nasal operation twenty years ago and a tonsil operation during childhood. Examination of the nose showed the septum to be thick and the nasal mucosa dry, with crusts on both sides. The right drum membrane was perforated and showed a thick scanty discharge. The left drum membrane was cloudy, somewhat retracted and movable. The patient received sixteen treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. The ear was irrigated with 33⅓ per cent alcohol. He reported that there was no longer any buzzing and that his hearing was somewhat better. Results determined objectively were: right ear—initial loss 38 decibels, terminal loss 34, gain 4; left ear—initial loss 35, terminal loss 34, gain 1.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Sept. 5, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on October 7.

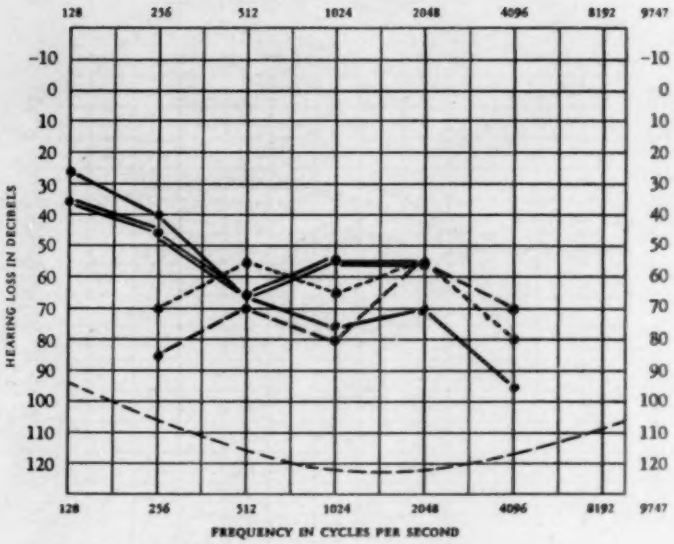

Chart 18 (case 18).—M. S., a white man aged 52, gave a history of deafness for the past forty-six years, since childhood, accompanied with noises in the ears. For a number of years he had had a discharge from the left ear, though the ear was dry at the time of my examination. The right drum membrane was markedly retracted; the left drum membrane showed a large central perforation, with healed scars over the rest of it. Examination of the nose revealed hypertrophy of the inferior turbinates and chronic rhinitis. There was no familial deafness. Twenty-eight treatments were given at the rate of three a week. These consisted of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. Subjectively the hearing and buzzing have not improved. Results noted objectively were: right ear—initial loss 54 decibels, terminal loss 55, loss 1; left ear—initial loss 37, terminal loss 32, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 6, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on March 27, 1940.

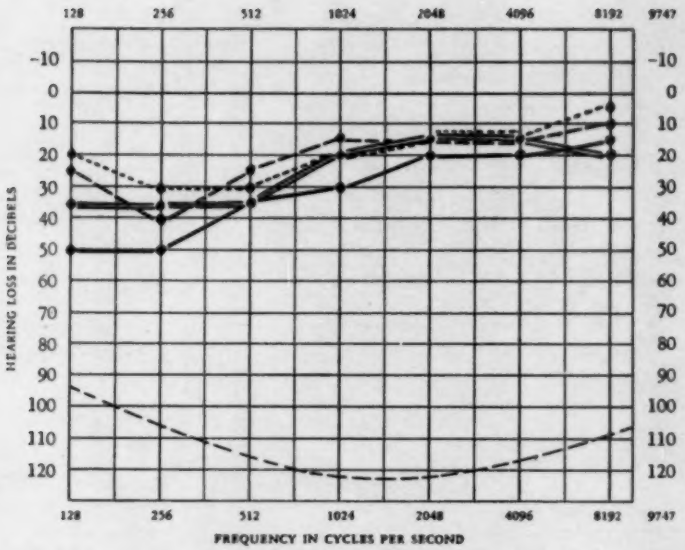

Chart 19 (case 19).—Mrs. I. F., a white woman aged 41, gave a history of loss of hearing for the past two years, with occasional buzzing. Examination of the ears showed both drum membranes to be retracted. Examination of the nose and throat revealed that the septum was deviated and that the tonsils were infected. A 10 per cent solution of mild protein silver was applied on nasal tampons, and a 1:2,000 solution of prostigmine methylsulfate was injected approximately three times a week until thirty treatments had been given. Subjectively the patient seemed to hear better, and the buzzing diminished. Results determined objectively were: right ear—initial loss 26 decibels, terminal loss 17.5, gain 8; left ear—initial loss 21, terminal loss 20, gain 1.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 20, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on July 15, 1940.

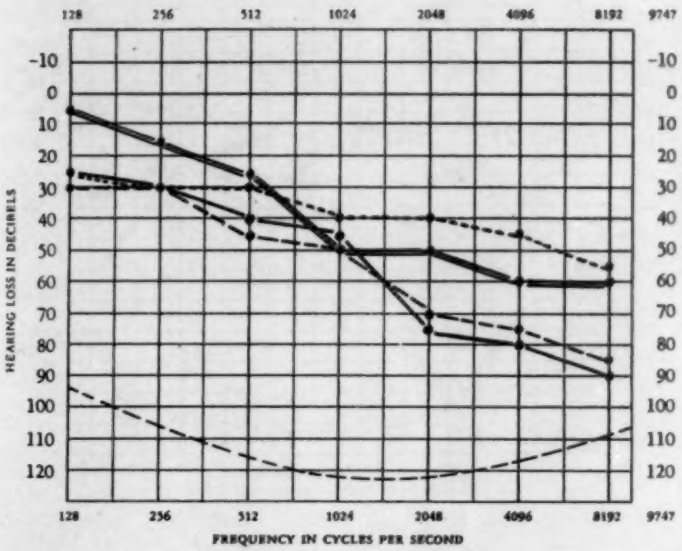

Chart 20 (case 20).—K. O., a white man aged 57, had had impaired hearing in both ears for the past three years. His ears had been catheterized about two years before he came for examination. He noticed occasional buzzing. Examination showed both drum membranes to be thickened and retracted. Twenty-seven treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate were given at the rate of three a week. On July 15, 1940 the patient stated that he had felt better until two weeks before. On September 11 his hearing was subjectively better. Results determined objectively were: right ear—initial loss 62.5 decibels, terminal loss 62.5, gain 0; left ear—initial loss 46, terminal loss 39, gain 7.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on April 10, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on September 11.

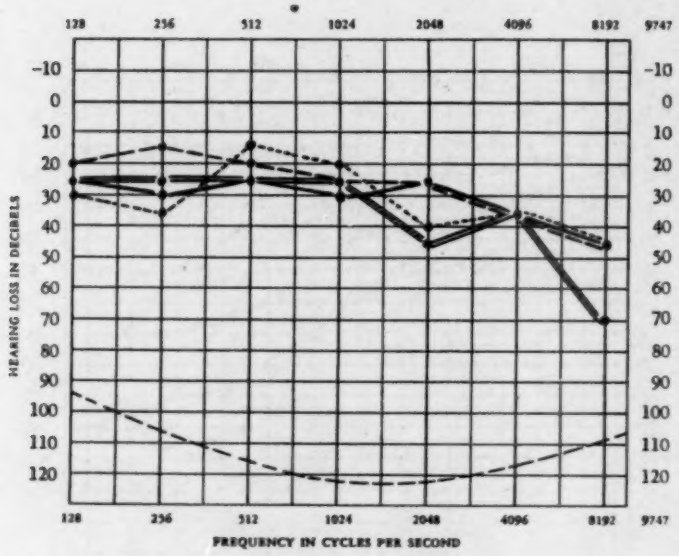

Chart 21 (case 21).—H. M., a white woman aged 60, had had buzzing in the left ear for the past six years. She did not complain of loss of hearing. Six months before she came for examination she experienced a feeling of dizziness after an attack of high blood pressure. This sensation had continued, with a feeling of falling. The past history and the family history were not significant. Examination showed that both drum membranes were cloudy over their entire extent and retracted. A diagnosis of Ménière's syndrome (?) was made. Nasal tampons saturated with a 10 per cent solution of mild protein silver were applied, and a 1:2,000 solution of prostigmine methylsulfate was injected three times a week until twelve treatments had been given. The patient remained away for one month. On July 15, 1940 she reported that the dizziness had cleared up and that she had been free from it until one week before. It had returned since, but in a much milder form. Subjectively her hearing seemed better than before. Results noted objectively were: right ear—initial loss 29 decibels, terminal loss 24, gain 5; left ear—initial loss 34, terminal loss 27.5, gain 6.5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on May 15, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on August 15.

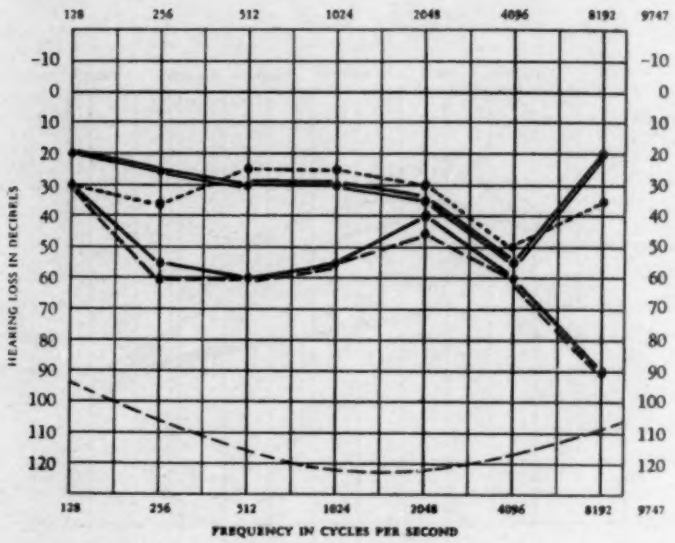

Chart 22 (case 22).—D. I., a white man aged 44, gave a history of some difficulty in hearing on the right for the past six to eight years. About four months previously he had contracted a cold, which lasted until two weeks before he came for examination. Since then, his hearing on the right had become markedly impaired. There was occasional buzzing in both ears. There was no family history of impaired hearing. His past history was noncontributory. Examination showed the right drum membrane to be slightly cloudy and retracted and the left one markedly so. Nasal tampons saturated with a 10 per cent solution of mild protein silver were applied and a 1:2,000 solution of prostigmine methylsulfate was injected three times a week until fifteen treatments had been given. Subjectively, the buzzing decreased markedly. At times the hearing on the right seemed better than it had before treatment, in clear weather. The hearing on the left became definitely better. Results determined objectively were: right ear—initial loss 29 decibels, terminal loss 26, gain 3; left ear—initial loss 32.5, terminal loss 27.5, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on May 15, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on September 11.

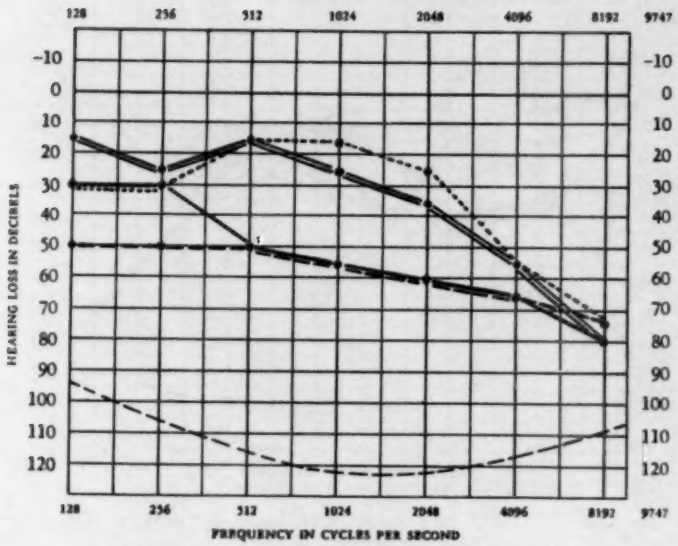

Chart 23 (case 23).—A. Mc., a white man aged 60, had not heard well since childhood and had been dizzy for the past six years. He had a discharge from the right nasal fossa. His past history revealed that he had had typhoid fever during childhood. Examination showed the right drum membrane to be retracted. The left drum membrane was normal. The Wassermann reaction was negative. Nasal tampons saturated with a 10 per cent solution of mild protein silver were applied, and an injection of a 1:2,000 solution of prostigmine methylsulfate was given three times a week until twenty-five treatments had been given. The dizziness remained the same as before treatment. Some improvement in hearing in the left ear was noted subjectively. Results determined objectively were: right ear—initial loss 57.5 decibels, terminal loss 57.5, gain 0; left ear—initial loss 32.5, terminal loss 27.5, gain 5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on April 24, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on July 15.

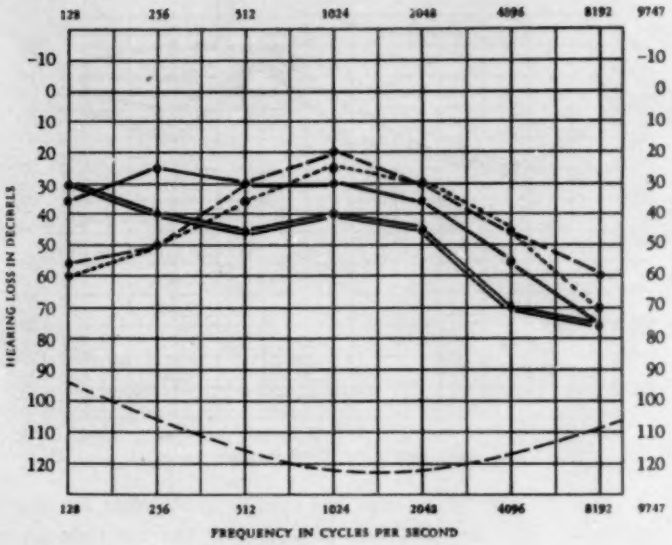

Chart 24 (case 24).—W. S., a white woman aged 50, gave a history of impaired hearing and buzzing of six years' duration, with rotary vertigo, frequent sore throat and complete anosmia on the right. Examination of the ears showed mild retraction of both drum membranes. The nasal septum was deviated to the left, and there was a polypoid right middle turbinate. The tonsils were diseased. Over a period of five months, thirty-six treatments were given, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. At the time of writing the patient reports that she can hear very well and has no tinnitus. Results determined objectively were: right ear—initial loss 37.5 decibels, terminal loss 31, gain 6.5; left ear—initial loss 50, terminal loss 34, gain 16.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 6, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on March 27, 1940.

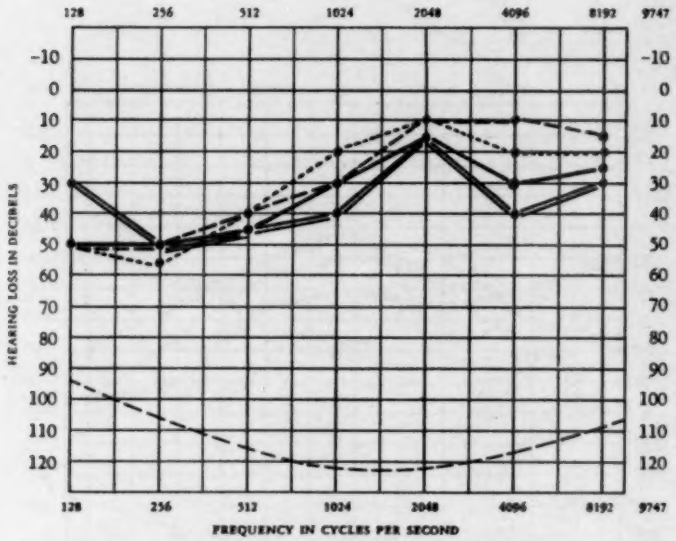

Chart 25 (case 25).—W. P., a white girl aged 9, was brought for examination. The mother claimed that the child had been hard of hearing since infancy and had had a nasal twang since childhood. The patient had a marked postnasal discharge. At the age of 1 year she had measles; at 2½ years, pneumonia with empyema on the right, which was drained; at 3½, a discharge from both ears, which continued for ten weeks, and a tonsillectomy; at 4, chickenpox. Examination of the ears showed that both drum membranes were retracted. There was a bilateral mucopurulent discharge from the nose. Roentgenograms of the sinuses, taken on two occasions, showed nothing abnormal. Forty-two treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:4,000 solution of prostigmine methylsulfate were given at the rate of about three a week. On Dec. 10, 1939 it was reported that the "teacher at school has noted marked improvement in hearing." On May 15, 1940, improvement in hearing was noted at home and at school. Results determined objectively were: right ear—initial loss 30 decibels, terminal loss 22.5, gain 7.5; left ear—initial loss 35, terminal loss 22.5, gain 12.5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 13, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on May 15, 1940.

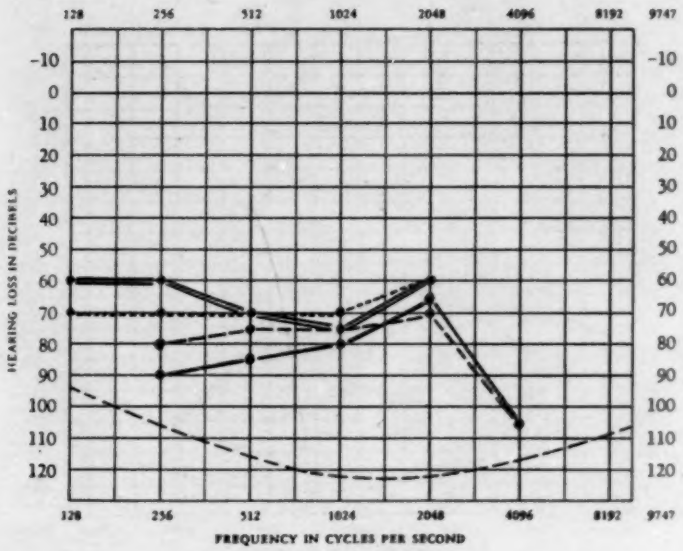

Chart 26 (case 26).—M. M., a white man aged 52, had been unable to hear on the right for the past twenty-five years. For the past ten months he had been unable to hear on the left. He had a constant hissing sound in the left ear and frequent colds in the nose. His past history revealed that he had had two nasal operations and a tonsillectomy. The mother was hard of hearing. On examination the right drum membrane showed marked retraction, a healed perforation and scars. The left drum membrane was retracted, with a large perforation, through which discharge could be seen. Examination of the nose revealed chronic atrophic rhinitis and chronic pharyngitis. Nine treatments, consisting of the application of nasal tampons saturated with a solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate were given at the rate of three a week. The patient did not return for treatment after Jan. 29, 1940. Results determined objectively were: right ear—initial loss 86 decibels, terminal loss 89, loss 3; left ear—initial loss 81, terminal loss 80, gain 1.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Dec. 27, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on Jan. 29, 1940.

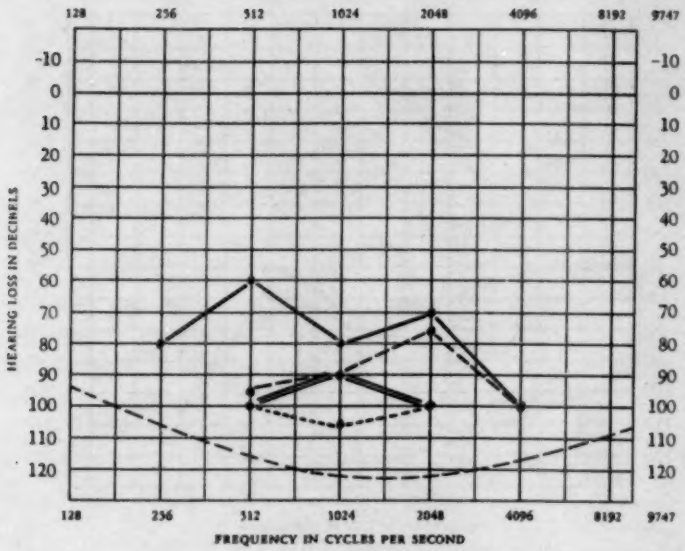

Chart 27 (case 27).—F. M., a white man aged 47, gave a history of deafness in the left ear since childhood. He had been hard of hearing in the right ear for the past few months and heard noises in both ears. One month before he came for examination, he had had an attack of dizziness. His past history was not significant. On examination of the ears, both drum membranes showed large perforations, with discharge. The nasal septum was deviated to the left. The pharynx was normal. The patient was given thirty-one treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. He complained of pain in the top of his head when he received the injections. The buzzing did not diminish. On March 19, 1940, he reported that his hearing was becoming worse. Results determined objectively were: right ear—initial loss 77.5 decibels, terminal loss 90, loss 22.5; left ear—initial loss 102.5, terminal loss 106, loss 3.5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 13, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on Feb. 19, 1940.

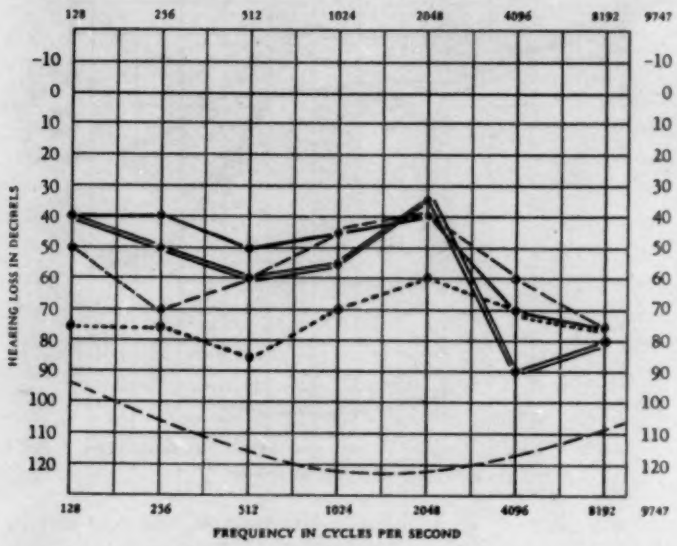

Chart 28 (case 28).—M. S., a white woman aged 41, gave a history of impaired hearing in both ears with tinnitus on the right, occasional headache, dizziness and vomiting. The hearing in the left ear had been impaired for one and one-half years; that in the right ear, for two months. Examination showed both drum membranes to be dull and markedly retracted. The nasal septum was deviated to the right and the turbinates were hypertrophied. The tonsils had been enucleated. Forty-one treatments, consisting of the application of nasal tampons saturated with a solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate, were given in about four months. On Nov. 20, 1939, the patient reported "vertigo and tinnitus practically gone" and said that she heard better. On March 27, 1940 she reported that the buzzing seemed to be more marked but that she could hear better over the telephone, even on a cloudy day. Results noted objectively were: right ear—initial loss 51 decibels, terminal loss 51, gain 0; left ear—initial loss 57.5, terminal loss 54, gain 3.5.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Nov. 15, 1939. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on March 27, 1940.

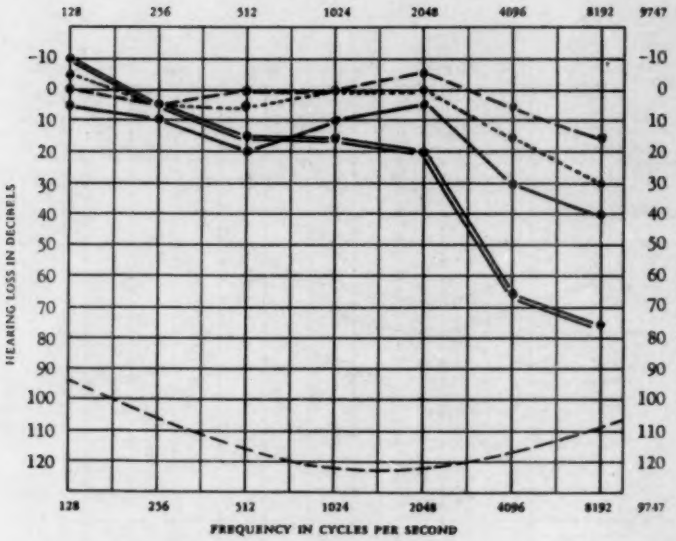

Chart 29 (case 29).—S. F. T., a white man aged 44, had been bathing in the ocean for the past few months. One week before he came for examination he had pain in the left ear, which cleared up without bleeding or discharge. Since then he had had complete loss of hearing on the left, with constant buzzing. He did not complain of the right ear, which, he said, is "normal." Seven years previously he had had a similar attack. Both ears were incised, and after a short time the condition cleared up. Examination showed the nasal septum to be deviated to the right, with mucoid discharge in both nares. The left drum membrane was markedly retracted, with somewhat hyperemic surface blood vessels, and movable. The right drum membrane was retracted and movable. The patient was given ten treatments, consisting of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver and the injection of a 1:2,000 solution of prostigmine methylsulfate. The left ear was catheterized. Subjectively, normal hearing returned to the left ear. At the time of writing, hearing on the right is better than before the treatments were started. No buzzing can be heard. Results noted objectively were: right ear—initial loss 16 decibels, terminal loss 0, gain 16; left ear—initial loss 29 decibels, terminal loss 5, gain 24. The improvement in hearing for the right ear should be noted, although that ear was not catheterized.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Aug. 5, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on September 6.

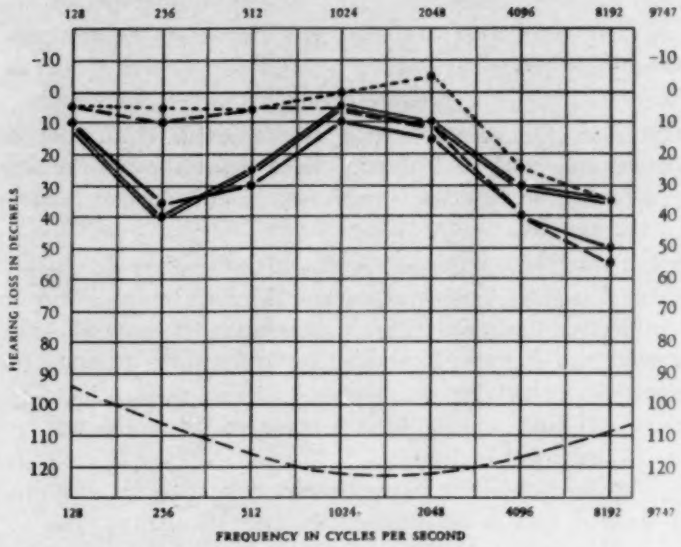

Chart 30 (case 30).—H. J., a white man aged 59, gave a history of tinnitus occurring at various intervals over a period of years. The condition had returned with great loudness and constancy in the last few months. He did not complain of any increased loss of hearing within the past ten years. There was no family history of impaired hearing. Two years before he came for examination, he had an attack of acute otitis media on the left. There had been a slight impairment of hearing over a period of many years, for which he received treatment at times. Examination showed both drum membranes gray, cloudy and somewhat retracted. The septum was deviated to the right. The tonsils were submerged. The patient was given fourteen treatments, consisting of the application of a solution of mild protein silver on nasal tampons and the injection of a 1:2,000 solution of prostigmine methylsulfate. Subjectively, it was noted that the buzzing abated at times and then returned. At the time of writing, the patient is pessimistic about the result of treatment. He is not aware of any loss of hearing. Results noted objectively were: right ear—initial loss 24 decibels, terminal loss 15, gain 9; left ear—initial loss 17.5, terminal loss 6.5, gain 11.

The black line indicates the audiometric reading for the right ear, and the double line the reading for the left ear, on Sept. 18, 1940. The dash line indicates the reading for the right ear, and the dotted line that for the left ear, on November 16.

Though the treatment and investigation covered approximately 100 cases, 30 cases are reported here. In the others the patients did not appear for treatment regularly, came for short intervals or were so erratic in their responses to audiometric checks as to make accurate evaluation impossible.

SUMMARY

Thirty cases of impaired hearing are reported. In 24 the impairment was chronic and in 1 acute. In 5 cases the involvement was considered recent, because the impairment of hearing had been present less than six months.

Nineteen patients complained of tinnitus aurium.

Nineteen patients, 6 with acute and 13 with chronic impairment, showed objective improvement of hearing; 16 showed subjective improvement, and 9 showed no improvement. Two patients reported that the condition had become worse.

Sixteen said that tinnitus had disappeared or diminished, while 3 reported no abatement of tinnitus.

Eleven showed objective improvement of hearing in all frequencies, while 3 showed loss in all frequencies and 16 showed loss in some frequencies and gain in others.

Improvement in Individual Tonal Frequencies

Frequency	Per Cent of Improvement in Ears Examined
512	56
1024	65
2048	51
4096	52

COMMENT

Loss of Hearing.—When there is a high decibel loss at the initial reading, an improvement up to 10 decibels does not evoke any subjective reaction and may not even be noted. When the initial loss is in the medium range, an improvement of even 5 decibels will elicit a subjective response. But the subjective response will show greater improvement on gain in some frequencies than on gain in others. The frequency of greatest value in this respect is 2048.

Of the 24 patients with chronic hearing impairment, 13 reported subjective improvement, while 19 showed some improvement on the chart.

All the patients with recent impairment (cases 3, 5, 11, 16, 21, 29), in none of whom the initial loss was greater than 35 decibels, reported definite improvement of hearing even though the greatest change in one ear was only 5 decibels (case 3). The patient in case 29 who did not

complain of any loss of hearing on the right and to whom no treatment was given, showed marked improvement.

Tinnitus.—Of the patients who complained of tinnitus, the 4 with recent impairment (cases 3, 5, 11, 16) and the 1 whose loss was acute (case 29) were relieved of this symptom completely. Of the 16 with chronic impairment who had this symptom, 11 reported improvement. Thus, one may assume that the use of prostigmine was of definite value. Patient 5, with some symptoms of Ménière's disease, reported complete recovery.

CONCLUSIONS

1. Prostigmine seems to have a definite value in impaired hearing of short duration and in some forms of tinnitus aurium.

2. Patients with chronic deafness show a smaller degree of improvement.

3. The patients whose cases are reported here do not show the high degree of improvement noted in the series presented by Davis and Rommel.

113 East Nineteenth Street.

CICATRICIAL ATRESIA OF THE ESOPHAGUS

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AND
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LOS ANGELES

Cicatricial atresia of the esophagus is rare. The most common single etiologic factor is ingestion of caustics, such as lye, washing soda, salts of tartar, household ammonia, mercury bichloride and phenol, either by accident or with suicidal intent. The other causes are systemic diseases involving the esophagus. Among them are scarlet fever, diphtheria, typhoid fever, pyogenic conditions, tuberculosis and syphilis. In a number of cases reported, one of the outstanding features was failure on the part of the patients to continue with the program of systematic dilation after the initial treatment. Instead, they waited complacently until the esophagus became completely impermeable.

The atresia may occur at any level, but most frequently it develops in the thoracic part of the esophagus where it is crossed by the left bronchus. The sites next in order, according to Jackson,[1] are the region of the cricopharyngeus muscle and the hiatal level The segment involved may be short or long. The shorter the stricture, the easier is its penetration. In the case reported by Kearney[2] the distance of obliteration was 6 cm., and both Jackson and Kearney thought it too hazardous to undertake endoesophageal dilation. The longest stricture reported to have been successfully treated was recorded by Mosher.[3] The length of obliteration was reported to be about 3 inches (7.5 cm.).

In 1935 Moersch[4] stated that among 310 patients with benign cicatricial stricture of the esophagus who were seen at the Mayo Clinic

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From the Bronchoesophagological Service, Department of Otolaryngology, College of Medical Evangelists.

1. Jackson, C.: Bronchoscopy, Esophagoscopy and Gastroscopy, ed. 3, Philadelphia, W. B. Saunders Company, 1934.

2. Kearney, H. L.: Cicatricial Atresia of Esophagus, Ann. Otol., Rhin. & Laryng. **44**:719 (Dec.) 1935.

3. Mosher, H. P.: The Use of the Esophagoscope in Esophageal Surgery, Boston M. & S. J. **165**:401 (Sept. 14) 1911.

4. Moersch, H. J.: Impermeable Cicatricial Stricture of Esophagus Treated by Modification of Iglauer Technic, Ann. Otol., Rhin. & Laryng. **44**:407 (June) 1935.

there were 17 who had impermeable stenosis. Heindl[5] reported that this complication occurred in 6 of the 137 patients observed from 1901 to 1925 at the Vienna Clinic.

The clinical picture of total atresia of the esophagus is characteristic. There are retention and regurgitation of all food ingested by mouth. The patient drools his saliva. To collect the oral secretion, he carries with him a pocket spit cup. When he is asleep in the recumbent position, the overflow from the esophagus tends to flood the lower air passages, with the possible danger of aspiration pneumonitis. Unless feeding through a gastrostomy tube is instituted, he is apt to lose weight rapidly and to have accompanying acidosis and dehydration. In cases in which the condition is long continued, a gastrostomy tube is invariably worn.

Fluoroscopy and roentgen examination with a contrast medium show a dilated atonic saclike structure, in the depth of which is the site of obstruction. If a colored fluid, such as a solution of methylthionine chloride U. S. P. (methylene blue), is taken by mouth, it fails to reach the gastrostomy opening. Chemical analysis of the gastric contents fails to indicate the presence of saliva.

REVIEW OF ENDOSCOPIC METHODS OF TREATMENT

Two types of operative procedure are used in the treatment of cicatricial atresia—plastic procedures,[6] which deal with the condition by an external approach, and the endoesophageal methods.

In this study we shall confine our attention to operative methods of the second type. Various procedures have been reported. Each appears to show certain advantages, but none is without danger.

The site of atresia may be exposed by

1. Peroral esophagoscopy.
2. Retrograde esophagoscopy with a smaller esophagoscope or with a cystoscope.
3. Combined peroral and retrograde esophagoscopy under monoplane or biplane fluoroscopic guidance.

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5. Heindl, A., Jr.: Klinische Beobachtungen an 137 gutartigen Osophagusstenosen der I. chir. Universitätsklinik Wien, 1901-1925, Deutsche Ztschr. f. Chir. **199**:252, 1926.

6. One of the earliest reported cases in which cervical esophagostomy was attempted for the relief of complete cicatricial atresia was excellently reported by Horsey. A child 5 years of age ingested lye, and he lived for almost two years. After bougienage failed, esophagostomy was done above the stricture. The atresia was firm and impassable with a bougie. Because of shock, the operation was discontinued. Death came in twenty-four hours. At autopsy the fibrous closure was located 4 inches (10 cm.) above the cardia. Horsey, C. W.: Stricture of the Oesophagus from the Action of Caustic Potash: Oesophagostomy; Death; Post-Mortem, Am. J. M. Sc. **72**:114 (July) 1876.

The stricture may be penetrated by

1. Electrolysis, diathermy, fulguration and electric cautery.
2. Forceps and metal olives for picking the involved site apart and ballooning by forcing air.
3. Catheterization either from above or from below, then retrograde dilation by means of the Tucker bougie.
4. Digital dilation of the cardia as an aid to instrumental puncture with the endoscope in situ.

Electrolysis.—Certain European workers, notably Guisez [7] and Heindl,[6] have selected electrolysis as the method of choice in the treatment of stricture of the esophagus. The method was first used in principle by E. Boeckel, in 1884. Later it was used by J. A. Fort and by Stroen. In Germany it was first used by Jeckels. It was introduced into Vienna by Lotheissen.[8]

Taking a cue from the urologic surgeon's use of a metal olive in circulatory electrolysis, a negative metal pole with a filiform conducting bougie on the tip of the long handle is introduced through an esophagoscope. The indifferent, or positive, electrode is placed on the chest or on the back. The strength of current used varies between 2 and 5 milliamperes. This treatment is applied weekly, alternating with simple dilations. Its purpose is to soften the cicatricial tissue by means of hyperemia and dampening.

Peroral Dilation—Ballooning and Picking of the Glued Area with an Olive-Tipped Dilator Followed by Introduction of a Soft Bougie from Above.—In 1914 Mosher [3] reported a case of corrosive stricture of a year's duration in a 5 year old boy. The stricture extended for a distance of 3 inches (7.5 cm.) in the midesophagus. The child had been fed through a gastrostomy tube. Before he came to Dr. Mosher's service, others had tried every manipulation, including the use of the cystoscope in the stomach and attack on the stricture from below, but had failed. At the first sitting Dr. Mosher employed a retrograde approach without success. He then utilized combined peroral and retrograde routes, and again failure resulted. He next attempted an approach from above, and he finally succeeded in reopening the passage by forcing air and picking the stricture apart with a small metal olive on a long handle followed by a soft bougie—a feat requiring much patience and dextral skill.

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7. Guisez, J.: De l'électrolyse circulaire endoscopique dans les sténoses cicatricielles graves de l'œsophage, Bull. et mém. Soc. de chir. de Paris **23**:266 (May 1) 1931; Rev. de laryng. **52**:377 (June 15) 1931.

8. Lotheissen, G.: Retrograde Gastroscopie und Oesophagoscopie, Wien. klin. Wchnschr. **46**:1317 (Nov. 3) 1933.

Peroral Penetration with Biting Forceps (Combined Peroral and Retrograde Esophagoscopy Under Guidance of Monoplane Fluoroscopy).—In 1924 King [9] made a comprehensive review of the literature concerning atresia of the esophagus and reported the case of a 24 year old woman in whom combined peroral and retrograde esophagoscopy was performed. Under monoplane fluoroscopic guidance, a long biting forceps was pushed downward through the strictured area into the lumen of the lower esophagoscope. This was followed by retrograde dilation. In 1926 Spickard [10] used a much similar technic. His patient was a woman aged 24 in whom cicatricial stricture developed after the ingestion of 2 tablets of mercury bichloride dissolved in 2 ounces (56 cc.) of saponated solution of cresol. A gastrostomy was done. Under monoplane fluoroscopic guidance, combined suprastricture and substricture esophagoscopy was performed. A long biting forceps was used through the upper endoscope, to reach the area of stricture, which appeared to be ½ to ¾ inch (1.25 to 2 cm.) thick. The forceps was passed by biting and pushing. This was followed by the introduction from above of filiform bougies and later by that of metal bougies. Spickard reviewed the case of stricture of the lower end of the esophagus reported by Taylor and another reported by Lange. In both cases the stricture was successfully dealt with by working from above with whalebone bougies. Taylor also cut the stricture with the Abbe string saw.

Combined Peroral and Retrograde Esophagoscopy (Stricture Approached from Above with the Filiform Bougie, under Guidance of Biplane Fluoroscopy).—Jackson [1] and his school have long advocated the use of biplane fluoroscopy in combination with peroral and retrograde esophagoscopy. They approach the stricture from above and dilate it with a filiform bougie. Jackson warns that "the penetrating part of this procedure is exceedingly dangerous unless the double-plane fluoroscope is used. Even then, it cannot be said to be free from danger." Jackson has treated several patients in this manner, with only one death.

In 1923 Clerf [11] reported the case of a 3½ year old boy, encountered at the bronchoscopic clinic in Jefferson Hospital. The site of the stricture was the thoracic part of the esophagus. The stricture was successfully penetrated by means of combined peroral and retrograde esophagoscopy, and then retrograde dilation was done, using the Tucker bougie. Unfortunately, measles developed and the child died of bron-

9. King, E.: New Method of Restoring Lumen in Case of Impermeable Stricture of Esophagus, Surg., Gynec. & Obst. **39**:596 (Nov.) 1924.

10. Spickard, P. W.: Strictures of Esophagus: Methods of Reconstruction of Esophageal Lumen, Northwest Med. **25**:90 (Feb.) 1926.

11. Clerf, L. H.: Cicatricial Stenosis of Esophagus Caused by Commercial Lye Preparations, J. A. M. A. **80**:1600 (June 2) 1923.

chopneumonia. Clerf [12] reported a case of cicatricial atresia in a child 10 years of age. He succeeded in passing the stricture from below with a filiform bougie and concluded that retrograde dilation by means of the Tucker bougie is the ideal method in the treatment of gastrostomized patients with cicatricial stenosis of the esophagus.

Combined Peroral and Retrograde Esophagoscopy (Electric Cautery Used from Above to Penetrate the Atresia).—Murphy and Iglauer [13] stated that they treated a patient with post-typhoid atresia of the thoracic esophagus by using an electric cautery from above for penetration of the glued area. Transillumination from the retrograde esophagoscope supplied sufficient light from above to guide the operator. Then the bougie was passed through the esophagoscope. After the operation the patient was able to swallow until his death, which occurred in three weeks and was thought to be due to mediastinitis.

Digital Manipulation and Retrograde Dilation.—When a stricture occurs at the cardiac end of the esophagus, the surgeon's index finger may be introduced through the gastrostomy opening. An esophagoscope is then introduced from above, directed toward the finger below, and the stricture may be penetrated by a suitable instrument.

In 1932 Wishart and Briggs [14] reported a case of complete atresia in a 40 year old man who accidentally swallowed sodium hydroxide. The stricture occurred 33.5 cm. from the upper incisor teeth. Gastrostomy was done. With the surgeon's index finger in the cardiac end of the stomach and the esophagoscope in the suprastrictured area, a long metal probe was passed through the esophagoscope and was made to pierce the center of the upper limit of the stricture. Then the probe was drawn down into the stomach. A string was drawn past the stricture and out through the original gastrostomy opening. Retrograde dilation was instituted, and a normal functioning of the esophagus was finally restored.

In 1935 Imperatori [15] recorded a case in which atresia occurred at the cardiac end of the esophagus. Dysphagia developed immediately after appendectomy. The operation was also complicated by subphrenic abscess. A gastrostomy and nine esophagoscopies were performed by other physicians before the patient consulted Imperatori, who successfully restored the lumen, using a technic similar to that described by Wishart and Briggs.

12. Clerf, L. H.: Atresia of the Esophagus, S. Clin. North America **4**:111 (Feb.) 1924.

13. Murphy, J. W., and Iglauer, S., in discussion on Jackson, C.: Esophageal Stenosis: Their Causes, Symptoms, Diagnosis and Treatment, Tr. Am. Laryng., Rhin. & Otol. Soc. **20**:211, 1914.

14. Wishart, J. N., and Briggs, W.: Complete Cicatricial Stricture of Esophagus: Case, J. Laryng. & Otol. **47**:464 (July) 1932.

15. Imperatori, C. J.: Complete Atresia of the Esophagus, Ann. Otol., Rhin. & Laryng. **40**:1194 (Dec.) 1931.

In 1936 Heatley [16] reported the case of a 47 year old patient who had intermittent dysphagia for 8 years. After the first dilation immediate relief was obtained. The patient was not seen again for two years, at the end of which time he returned because of complete obstruction. Gastrostomy was done. A ureteral catheter failed to penetrate the stricture. An esophagoscope was then lowered to the point of obstruction. A small metal dilator was gently forced up through the strictures until it appeared in the esophagoscope. This was followed by the introduction of a ureteral catheter, which was drawn up to the mouth, and a continuous circle of string was established for dilation.

Use of a Cystoscope in the Cardia, Followed by Dilation Either from Above or from Below.—The use of the cystoscope antedates the invention of the esophagoscope. In 1868 Kussmaul used a Desormeaux urethroscope elongated to 43 cm. for direct examination of carcinoma of the thoracic part of the esophagus. In the case of the patient who was successfully treated by Mosher, a cystoscope had been first used in the stomach for retrograde esophagoscopy by another physician but had failed to aid in relieving the atresia. Iglauer,[17] in 1932, and Carroll and Myers,[18] in 1937, advocated the use of the cystoscope as an instrument of retrograde esophagoscopy, to be followed by the introduction of a urethral catheter from below. In 1935 Moersch [4] modified the technic advocated by Iglauer. An olive-tipped no. 8 leaded catheter was threaded in a small caliber hollow tube. This was placed within an esophagoscope. Under guidance, it gradually forced the stricture apart until it reached the surgeon's hand in the stomach. A thread was tied to the catheter and withdrawn out of the mouth, making a continuous circle for dilation.

There are several advantages in using the cystoscope for retrograde esophagoscopy. When the stomach is thoroughly washed and filled with sterile water through the gastrostomy wound, the cystoscope reveals a much wider area and gives a better view of the field than can be obtained through an open tube esophagoscope. Because of its smaller size the cystoscope can be insinuated into the esophagus readily under visual guidance without trauma to the delicate structures. In the case of a permeable stricture, a ureteral catheter can be insinuated upward much more easily than esophageal bougies can be inserted from above downward. When atresia of the esophagus is present, with its usual sac-like dilation of the upper segment, the thicker and smoother walls and

16. Heatley, C. A.: Some Problems in Esophageal Atresia, Ann. Otol., Rhin. & Laryng. **45**:1122 (Dec.) 1936.

17. Iglauer, S.: Impermeable Stricture of the Esophagus Relieved by Retrograde Bougienage with the Aid of a Cystoscope Inserted Through a Gastrostomy, Ann. Otol., Rhin. & Laryng. **41**:1191 (Dec.) 1932.

18. Carroll, G., and Myers, E. L.: Use of Cystoscope in Impassable Esophageal Stricture, Urol. & Cutan. Rev. **41**:96 (Feb.) 1937.

more axial approach from below render the penetration much safer and more accurate than when the approach is attempted from above. In the case reported here, we found the cystoscope admirably adapted as an instrument of retrograde esophagoscopy.

REPORT OF A CASE

On July 14, 1938, J. M., a Mexican woman aged 24 years, was admitted to the surgical service of the White Memorial Clinic. Her chief complaints were intermittent pain and bleeding of five months' duration about her gastrostomy tube, which she had worn for the previous five years. She had been nauseated and had had attacks of vomiting for one month. On July 17 she was referred to the bronchoesophagologic service for consultation and treatment.

History of Illness.—On July 12, 1930, while working in a maternity hospital in Arizona, she accidentally swallowed a cup of sodium hydroxide. She was then 16 years of age. Her health had been perfect and she weighed 140 pounds (63.5 Kg.). Emergency treatment was given by the local physician, and she was sent to her country home, where her mother was to care for her. She was able to swallow nothing but liquids, and later, when the symptom of dysphagia persisted, she was fed eggnog by retention enema. She stated that her weight went down to 50 pounds (22.5 Kg.) in six weeks. (The loss of 90 pounds [41 Kg.] was questioned, but the patient reaffirmed it.)

On September 9 Dr. R. B. Raney, of Phoenix, Ariz., performed a gastrostomy. After this operation the patient regained her strength and much of her weight. In December 1930, under the direction of another group of surgeons, a program of peroral esophagoscopic dilation was instituted, and she was able to take liquids. In June 1931 her gastrostomy wound was closed. She weighed 97 pounds (44 Kg.). Shortly after this she began to suffer from progressively increasing dysphagia, and by September 1931 she was no longer able to take even liquids by mouth. The gastrostomy wound was reopened. Efforts to reestablish the esophageal channel were continued by a weekly dilation until some time in 1932, but she failed to respond to the treatments. For a year no medical attention was given. She continued to be fed through the gastrostomy tube.

In 1935 a third group of surgeons attempted a retrograde esophagoscopic dilation but failed. After the manipulation, a septic type of temperature developed and she suffered considerable pain in the chest. She remained in the hospital for six weeks, at the end of which time she was dismissed without any improvement in her swallowing. Prior to January 1938 she had consulted eighteen physicians. Numerous roentgenograms had been made of her esophagus, but the condition was considered an inoperable atresia, and the patient had fully resigned herself to the fate of carrying a pocket spit cup and taking feedings through a gastrostomy tube.

Physical Examination.—The patient was a well developed, fairly well nourished, mentally alert young Mexican woman, in no apparent pain or distrees. Her blood and urine were normal. The Wassermann reaction of the blood was negative. Roentgen examination of her esophagus made with the aid of barium sulfate indicated complete obstruction in the middle third.

On Aug. 2, 1938 peroral esophagoscopy was performed. A dense stricture involving the area revealed in the roentgenogram was found (figure). Above this segment the esophagus was greatly dilated. In the bottom of this sac, slightly

to the right, was a small dimpled pocket. A soft filiform bougie was used to locate the channel, but further passage was not found. Ballooning by air was equally unsuccessful. On August 26 Dr. Roger Barnes, of the genitourinary department, passed a McCarthy no. 20 panendoscope through the gastrostomy opening and introduced a ureteral catheter into the esophagus below the stricture. Then 10 cc. of skiodan, a radio-opaque dye, was introduced. A barium sulfate meal was given by mouth. Roentgenograms indicated the extent of the stricture to be about ½ to ¾ inch (1.25 to 1.9 cm.).

Treatment.—On Sept. 2 the patient was taken to the fluoroscopic room, and as in the previous retrograde esophagoscopy, a panendoscope was insinuated into the lower end of the esophagus. The site of the stricture was well visualized. Through the cystoscope a fulgurating tip was inserted against the partition of the stricture.

Retouched photograph, showing barium sulfate in the upper segment of the esophagus and skiodan introduced through a catheter in the lower segment. The length of the stricture appeared to be ½ to ¾ of an inch (1.25 to 2 cm.).

A Jackson 10: 45 full lumen esophagoscope was then inserted from above against the obstructing area. The two instruments met 25 cm. from the upper dental arch. As viewed through a double plane fluoroscope, the fulgurating tip appeared to point too far posteriorly. A normal no. 8 ureteral catheter was substituted. The catheter contained a wire stylet, the distal end of which projected 1 cm. beyond the tip of the catheter. This was placed against the substrictural surface. The wire was connected with the cutting current originating in a diathermy instrument. Again, under fluoroscopic guidance the catheter from below and a metal probe through the esophagoscope from above were placed in axial line. The cutting current was applied, and it fulgurated through the scarred partition.

At this stage the catheter was pushed upward and grasped by forceps in the hand of the peroral esophagoscopist. The cystoscope was removed. A thread

was tied on the distal end of the catheter. This was pulled upward through the esophagoscope. The upper end of the thread was passed through the nostril and was anchored to the cheek, the other end being left in the stomach. The patient reacted well to the surgical treatment and was dismissed from the hospital in two days. On September 7 (five days later) a program of weekly retrograde esophageal dilations was instituted. Tucker's continuous end to end bougienage was used. By the end of October the woman was able to swallow all kinds of food. Dilation was continued until the summer of 1939. Sept. 5, 1939, a year after the reestablishment of the esophageal channel, she was readmitted to the hospital, and Dr. Clarence E. Stafford, of the surgical department, closed the gastrostomy opening. On September 19 she was dismissed in good condition. She has returned to the clinic several times since the operation. She has been completely free from esophageal symptoms and is living a normal healthful life.

An interesting historical side light is that one or two years prior to her coming to our clinic the patient had married a Mexican who prenuptially was not aware of her esophageal occlusion or that she fed herself through a gastrostomy tube. He reacted chivalrously to the revelation and made up his mind to do all in his power to effect relief of her distressed state. His aim has been accomplished.

COMMENT

The treatment of total atresia of the esophagus is extremely difficult and fraught with grave consequences (Vinson [19] and Kearney [2]). The fact that various procedures are being advocated and practiced by each operator is indicative of the necessity for individual treatment of each patient. When the atresia occurs at the lower end of the esophagus, bimanual dilation appears to be the operation of choice. In those cases in which the thoracic part of the esophagus is involved, combined peroral and retrograde esophagoscopy under monoplane or biplane fluoroscopic guidance facilitates attack on the glued area either from above or from below. To us the substrictural attack seems to offer a greater margin of safety, since the walls of the esophagus are thicker in the lower segment, the approach is more axial, and the instruments are less likely to invade the mediastinum.

SUMMARY

A case of complete cicatricial atresia of the esophagus of six years' duration is reported. A normally functioning esophageal lumen was established by endoesophageal means.

Miss L. Marguerite Prime, of the library and the department of literary research of the American College of Surgeons, Chicago, and Mrs. Ella Moyers, librarian in charge, White Memorial Hospital, Los Angeles, aided in the work.

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19. Vinson, P. P.: Cicatricial (Benign) Stricture of Esophagus: Tabulated Report of One Hundred and Eighty-Six Cases, Ann. Otol., Rhin. & Laryng. **36**: 40 (March) 1927.

Case Reports

OTOSCLEROSIS IN IDENTICAL TWINS

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Otosclerosis is still one of the important unsolved problems of otology, and any light which may be thrown on the subject is of value. Because of the tendency of the condition to occur in members of the same family, a study of the family of a person with otosclerosis has always been of interest. When, in addition, one can study a pair of identical twins suffering from otosclerosis, there is hope that something new may be learned.

Few cases of otosclerosis in identical twins have been reported. The most extensive article on the subject is that of Shambaugh Jr.,[1] who reported 3 instances of otosclerosis in identical twins. Shambaugh and Shambaugh[2] had previously reported 2 instances. Shambaugh Jr.[1] stated that the first report of the condition in identical twins was that by Albrecht in 1932, and the second that by Rodin in 1933. Twice he has found that the disease ran an identical course in each twin. In 2 other instances the disease began five years earlier in the twin who was the more susceptible to head colds. In another the deafness began sixteen years earlier in one twin. The most likely cause for the striking difference was the much greater susceptibility of that twin to head colds. Fowler[3] also expressed the belief that head colds and the development of otosclerosis are related. In discussing otosclerosis Selfridge[4] stated that he suspected a faulty diet as the cause, i. e., one especially low in citrus fruits, milk and eggs. Bunch[5] has noted that persons with otosclerosis have low basal metabolic rates and soft teeth.

The following is a record of an instance of otosclerosis in identical twins:

REPORT OF A CASE

Jean and Florence, aged 24, were first examined in 1938. Jean came for examination complaining of deafness, and, because of the findings and interesting history, her twin sister and also their mother, aged 43, were examined.

The mother's history revealed that she was 1 of 10 children. She had 3 brothers and 6 sisters. Of the sisters, Marie, aged 41, has been deaf since child-

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1. Shambaugh, G. E., Jr.: Otosclerosis in Twins, J. A. M. A. **104**:1216 (April 6) 1935.

2. Shambaugh, G. E., and Shambaugh, G. E., Jr.: Progressive Deafness in Identical Twins, Arch. Otolaryng. **17**:171 (Feb.) 1933.

3. Fowler, E. P.: Otosclerosis Complicated by Other Lesions: A Study of Roentgenograms, Audiograms, Laboratory and Clinical Findings, Ann. Otol., Rhin. & Laryng. **42**:714, 1933.

4. Selfridge, G.: Chronic Progressive Deafness, Mid-Winter Clinical Course, Los Angeles, January 1938.

5. Bunch, C. C.: Some Observations on Clinical Otosclerosis, Ann. Otol., Rhin. & Laryng. **43**:344, 1934.

hood; Margaret, aged 47, has normal hearing; Minkus, aged 51, has noticed deafness the past few years; Nellie, aged 53, who died in 1936, had been deaf since childhood; Drina, aged 55, was just beginning to notice deafness, while Kay, aged 53, who died in 1930, had been deaf since she was a child. Of the 3 brothers, Jack, aged 45, had noted deafness starting when he was 11 years old; Henry, aged 47, at the time of his death, in 1936, had normal hearing, while Harry, aged 53, has had deafness since he was a boy. The only one of these examined by me was Harry. On examination, in 1932, his right drum membrane was acutely inflamed and the ear discharging. This condition followed swimming and diving. The left drum membrane revealed a healed perforation. Because of the otitis media, a diagnosis of otosclerosis could not be made, although deafness had been present for many years. None of the other siblings gave a history of ever having had a discharging ear.

The twins' mother stated that she noticed increasing deafness in the left ear at about 17 years of age. Hearing was better over the telephone. At the age of 20 years she married and at 21 gave birth to Jean and Florence. The patient was delivered by Dr. G. J. Hagens, and the hospital record states that the placenta was delivered spontaneously. It was larger than a single placenta, of normal shape, with lateral cord insertions. The membranes were thin, complete and with two compartments. Anesthesia was induced with chloroform and 1 cc. of a solution of posterior pituitary U. S. P. was given just before delivery and 1 drachm (3.88 Gm.) of fluidextract of ergot afterward. The mother's health prior to and during pregnancy was good except for a moderate pyelitis of the right kidney the last week of pregnancy.

A few days after the delivery, increased deafness, especially in the right ear, was noted. The loss of hearing has slowly progressed to the time of writing. Tinnitus has occurred intermittently. Removal of the tonsils five years ago made no change in the hearing; neither did a series of inflations. Recently, she has taken a preparation of vitamin B complex but without noticeable results. At the time of writing the patient hears well with a bone conduction hearing aid. The birth of a boy seven years after that of the twins apparently did not influence the deafness appreciably. To date, this child hears normally. Examination of the drum membranes of the mother showed some thickening. Tuning fork tests revealed a negative Rinne reaction, no shortening of bone conduction and marked impairment of both lower and upper tone limits. Loud voice was heard at each ear. An audiogram (fig. 1 *A*) shows the condition of her hearing in March 1938. A diagnosis of otosclerosis was made. As far as can be ascertained, the patient's husband has normal hearing.

At the time of her examination, in January 1938, Jean, the first twin examined, had noticed deafness for four years. The left ear was involved at the onset and the right one later on. There never had been any discharge, pain or tinnitus. Jean was married when she was 17 years old, about the time her deafness began. Soon after the birth of a child, one year later, the hearing became worse. Examination of Jean showed the drum membranes to be essentially normal; the Weber test showed lateralization to the left; the Rinne reaction was negative in both ears; the lower tones were not heard and high tones were somewhat diminished; bone conduction was not shortened, while the whispered voice was not heard on the left side but was heard at 2 to 3 feet (61 to 91 cm.) on the right. An audiogram, made in July 1938, is shown in figure 1 *B*. Otosclerosis was considered to be the cause of deafness.

Florence noted beginning deafness some time after her sister did. The hearing defect has not seemed severe enough for her to seek medical aid. The right ear

was first involved and the left one more recently. Examination revealed normal drum membranes, with a suggestion of Schwartze's sign on the right side. Hearing tests showed a negative Rinne reaction, no shortening of bone conduction, elevation of the lower tone limit on the right side and a slight defect in hearing for the upper tones, while the whispered voice was heard at 3 to 6 inches (7.5 to 15 cm.) on the right and at 3 to 6 feet (91.5 to 183 cm.) on the left. The audiogram (fig. 1 *C*) made in March 1938 showed definitely better hearing than that recorded for Jean. The eustachian tubes inflated easily but without producing improvement in hearing. A diagnosis of otosclerosis was made.

A comparison of the twin girls with respect to development and environment is of interest. The sisters have always been difficult to tell apart (fig. 2). When

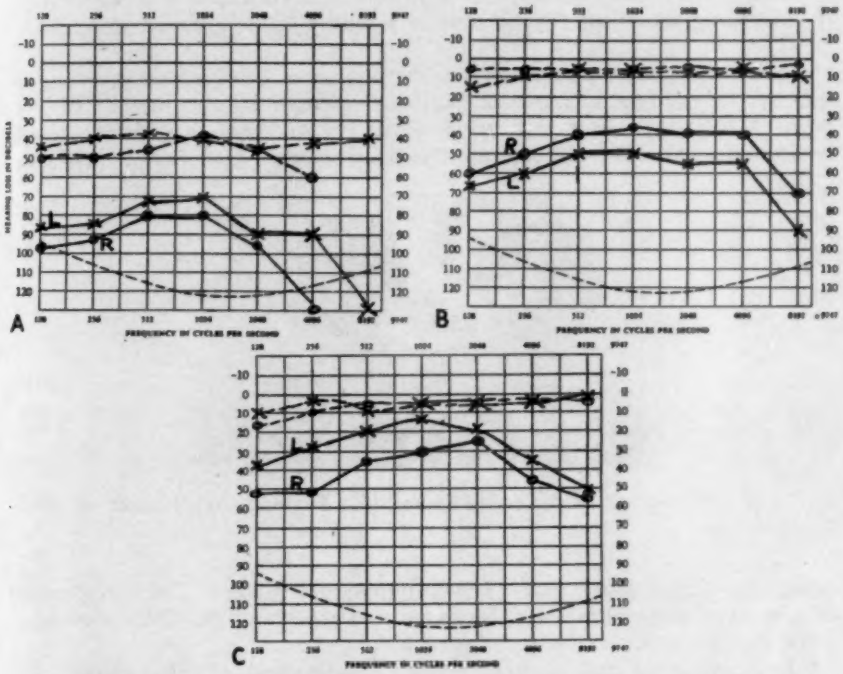

Fig. 1.—The solid line indicates air conduction and the dotted line bone conduction. *o* is right; *x* is left. *A*, audiogram of Mrs. R., the twins' mother, March 1938. *B*, audiogram of Jean, July 1938. *C*, audiogram of Florence, March 1938.

they were young, the difference in their voices was used by the relatives to distinguish them. They were reared together and attended the same schools, being in the same grades throughout. They had measles and whooping cough and were about equally ill. Neither has had scarlet fever, diphtheria or mumps. Both have had poor teeth, which have crumbled away without much evidence of infection, and at the time of writing are wearing upper plates. Jean (married) has had her plate since she was 19, while Florence procured hers six months later. As children the girls were fond of sweets, and their diet consisted mainly of starches and sugars. In common, they have frequent colds, and their mother has this history also. In spite of the colds, neither girl has had a known aural infection

or discharge. In 1936 Jean had her tonsils removed because of quinsy. Florence has small, slightly reddened tonsils. She also has had quinsy in the past. Fingerprints of the twins taken recently show, according to Dr. W. M. Krogman, of the University of Chicago, sufficient evidence to corroborate the statement that the twins are identical.

COMMENT

This report illustrates the occurrence of deafness in many members of a family. Whether otosclerosis has been present in all of these persons cannot be determined, as sufficient data are lacking. In Harry, examined by me, there is definite evidence only of otitis media, but otosclerosis might be present and masked by the otitis. Study of temporal bones post mortem has revealed examples of just such a situation. In any event it is striking that 8 of 10 children in a family have suffered from deafness.

The influence of pregnancy on the course of otosclerosis is brought out by the definite increase in deafness suffered by the mother of the twins a few days after they were born. When Jean was delivered of her child, she also noticed increasing deafness, and her hearing is

Fig. 2.—Photograph of the twins (Jean, left; Florence, right) taken at about 1 year of age.

definitely worse than that of her unmarried sister. The pregnancy of Jean is of unusual interest, because in all aspects of childhood development and experience the girls were alike.

In support of the suggestions of Fowler and of Shambaugh Jr. regarding a history of frequent colds, both the mother and the twins gave such a history. Selfridge expressed a suspicion that faulty diet is a cause of otosclerosis, and these twins were known to eat an excess of starches and sugar. They also disliked fruits and vegetables, although they drank plenty of milk. The fact that their teeth disintegrated early coincides with the experience of Bunch. However, this condition existed in most of the members of the family on the father's side, and yet none have had deafness. These highly suggestive points are presented for what they may be worth. It must be borne in mind that the factors referred to are present in many cases in which there are no manifestations of clinical otosclerosis. It is possible, as Fowler has suggested, that routine examination of temporal bones might reveal many instances of otosclerosis in which the focus did not involve the footplate and hence did not produce deafness. My own work with temporal bones bears

this out. Therefore, until more routine examination of temporal bones is done, it will not be known how many cases of otosclerosis have been missed, in which some of these factors may have been at work. For the present it is probably best to place on record any information which may throw light on this unsolved condition.

SUMMARY

A case of deafness in identical twins is reported, in which the deafness of one twin has increased over that of the other. The only known factor to account for this is that the twin with the poorer hearing is married and has had a child. Her hearing became noticeably worse when the baby was born and has remained poorer than her sister's.

30 North Michigan Avenue.

CHRONIC MASTOIDITIS, MENINGITIS AND LABYRINTHITIS; OPERATION; RECOVERY

JACKSON A. SEWARD, M.D., NEW YORK

Since sulfanilamide has been made available to the members of the medical profession, many cases have been reported in which otitic meningitis has been cured. It is such a case that I wish to review here.

A white man aged 57, of Polish nativity, a waiter, was admitted to the ear, nose and throat service of Bellevue Hospital, New York, on May 21, 1940. The chief complaint was pain of five days' duration in the left temporal region. Six months prior to admission there was a spontaneous rupture of the drum membrane of the left ear, and the discharge had been continuous since that time, with a considerable increase in amount shortly before the patient entered the hospital. One week prior to admission, hearing in the left ear became much impaired. There was no vertigo, nausea, vomiting or spontaneous nystagmus. The right drum membrane was retracted; the left had a perforation in the posterior inferior quadrant, with a small amount of brown, putrid, purulent discharge. The posterior superior part of the canal was sagging, and there was tenderness over the mastoid antrum. The static labyrinths responded normally to the caloric test. According to the 2-A audiometer test, there was a hearing loss in the critical frequencies of 34 decibels in the right ear and 50 decibels in the left ear. Roentgenograms showed the mastoids to be pneumatic in type, the right clear and the left hazy throughout. A smear of the aural discharge showed gram-negative bacilli, and a culture showed Staphylococcus albus, Bacillus proteus and Streptococcus viridans. The Wassermann reaction was negative, and the blood count was normal. A diagnosis of chronic mastoiditis on the left was made, and the patient was kept under observation and treatment for one week, during which time his temperature was normal but the ear showed no improvement.

On May 27 the patient was examined by Dr. J. Winston Fowlkes, the chief surgeon, who advised a radical mastoidectomy on the left side. On the following day this was done by another member of the staff. Considerable necrosis of bone was found in the mastoid antrum and middle ear. The facial nerve was exposed during this operation, and facial paralysis on the left side was evident the next day. Each day during the first week after operation there was a slight elevation of temperature, but only once was it more than 1 degree. During the next nine days the temperature fluctuated between the normal and 102.8 F. On the seventeenth day after operation it reached 103.2 F. and the patient complained of pain in the left side of his head and the back of his neck. The mastoidectomy cavity appeared to be healing nicely. There was no complaint of vertigo at this time, and there was no spontaneous nystagmus. However, in the nurse's notes it was found that three days before, which was the fourteenth day after operation, the patient had complained of dizziness and of pain in the left side of his face. On the morning of the eighteenth postoperative day he was drowsy and had a stiff neck. A spinal tap was done. The fluid was under increased pressure, was cloudy

Read before the Section of Otolaryngology of the New York Academy of Medicine on Jan. 15, 1941.

and contained 98 per cent polymorphonuclear cells. The total cell count reported was too high for me to believe. There must have been an error in technic. A smear of the fluid showed cocci, and a culture, reported later, showed hemolytic streptococci. At 2 p. m., when I saw the patient, he was lying on his right side with his eyes closed and appeared very drowsy. The temperature was 103.6 F. and the pulse rate 96. His neck was stiff, and Kernig's sign was present. I was unable to determine whether or not there was spontaneous nystagmus. Immediate operation was advised, and an extensive exposure of the dura in the middle and posterior fossae was made. The dura in the posterior fossa was opened. Having found no localized pus thus far, I felt justified in opening the labyrinth. The horizontal canal was opened first and then the cochlea. Pus was found in both. A large quantity of cerebrospinal fluid escaped through the cochlea and continued to drain for ten days. Administration of sulfanilamide, 1.3 Gm. every four hours, was ordered. On the third day this was reduced to 1 Gm. every four hours and on the seventh day to 0.3 Gm. every four hours. The temperature gradually receded, and, on the fifth day after the labyrinth was drained, it became normal and remained so. On the sixth day the neck was not stiff and Kernig's sign was not present. At the height of the meningitis, the white blood cell count was 28,450 per cubic millimeter, with 88 per cent polymorphonuclears and 12 per cent lymphocytes. Frequent counts were made, and on the eleventh day the white blood cell count was normal and the red blood cells numbered 3,200,000. One week after administration of sulfanilamide was stopped, this slight anemia had entirely disappeared. The patient received a total of approximately 60 Gm. of sulfanilamide within two weeks. On the twenty-first day after the operation on the labyrinth he was up in a wheel chair, and on the following day he was walking about the ward.

I saw him on Jan. 2, 1941. He stated that except for an occasional mild attack of dizziness of short duration and a slight pain in the left side of his head he felt well. The cavity from the radical operation was dry and there was a total loss of hearing in the left ear in addition to facial paralysis on the left side.

COMMENT

This was evidently a case of latent purulent labyrinthitis. The condition gave few symptoms and virtually no signs and, with little warning, released a shower of pus on the meninges.

Bowers [1] stated that the operative treatment of meningitis demands early recognition of meningeal irritation, prompt surgical removal of the focus and treatment with sulfanilamide. In this case the labyrinth was drained and administration of sulfanilamide started less than twelve hours after definite signs of meningitis were observed.

Cinelli [2] said that labyrinthitis coming on in the third week of acute purulent otitis media or later is usually of a purulent type. On the fourteenth day after radical mastoidectomy this patient complained of vertigo, and on the eighteenth day he had definite signs of meningitis.

1. Bowers, W. C.: Operative Treatment of Meningitis, Laryngoscope **49**: 26-29 (Jan.) 1939.

2. Cinelli, A. A.: Clinical Forms of Labyrinthitis, Laryngoscope **48**:157-167 (March) 1938.

Dench,[3] Smith,[4] Eagleton [5] and others have stated that the labyrinth is the most common pathway of infection from the ear to the meninges.

Friesner and Rosenwasser [6] said they had found suppuration of the petrous pyramid to be the most common pathway of extension to the meninges.

Kopetzky [7] held that infections usually follow the route of the blood vessels rather than the preformed anatomic channels.

Jones [8] stated that in patients who have died of meningitis the more common avenues of infection were the natural channels through the bone, such as the ductus cochlearis and ductus endolymphaticus. He mentioned two rather unusual routes, namely, that through the carotid canal and that along the facial nerve.

Appelbaum [9] said that the maximum concentration of sulfanilamide in the blood is reached four to six hours after the drug is given and that the drug rapidly passes over into all body fluids, including the spinal fluid, in approximately the same concentration as that in which it is found in the blood. He also said small doses at frequent intervals are more effective than large doses at longer intervals.

All the authors I have mentioned recommended prompt removal of the primary focus. In the case reported here, sulfanilamide undoubtedly played a large part in the cure of the septic meningitis. However, I feel that prompt drainage of the labyrinth immediately after meningeal signs became evident contributed in no small way to the patient's recovery.

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3. Dench, E. B.: Treatment of Acute Otitic Meningitis, Am. J. M. Sc. **139**: 157-165 (Feb.) 1910.

4. Smith, S. M.: The Indications for Surgical Interference for Relief of Otitic Meningitis, J. A. M. A. **55**:757-759 (Aug. 27) 1910.

5. Eagleton, W. P.: Paths of Infection in Otitic Meningitis, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 601.

6. Friesner, I., and Rosenwasser, H.: Surgical Treatment of Otitic Meningitis, Laryngoscope **48**:472-479 (July) 1938.

7. Kopetzky, S. J.: Management and Treatment of Otogenic Meningitis, Ann. Otol., Rhin. & Laryng. **47**:117-134 (March) 1938.

8. Jones, M. F.: Avenues of Infection in Otitic Meningitis, Laryngoscope **49**: 23-25 (Jan.) 1939.

9. Appelbaum, E.: Sulfanilamide and Related Compounds in Treatment of Otitic Meningitis, Laryngoscope **48**:482-498 (July) 1938.

ESOPHAGEAL FOREIGN BODY WITH SYMPTOMS SUGGESTING OTHER THORACIC DISEASE

STANTON A. FRIEDBERG, M.D., CHICAGO

This report presents an unusual group of symptoms caused by a foreign body in the esophagus and the difficulties attendant on establishing a correct diagnosis. The problem was further complicated by the clinical conditions simulated, which, if present would have constituted a definite contraindication to any endoscopic procedures.

REPORT OF A CASE

Mrs. M. H., aged 35, experienced a sudden severe pain in the chest, beneath the sternum, while eating soup. She had no knowledge of having swallowed a foreign body and no sensation of any object having lodged even temporarily in the throat. The soup had been made by boiling beans and spareribs. The patient denied the possibility that the ribs had been splintered previously or had disintegrated as a result of the boiling. There was no dysphagia or pain on swallowing.

The pain in the chest was described as penetrating and constant, with localization in the midline and radiation to the back. No pains in the shoulder or arms were noted, nor could a previous history of any comparable symptoms be elicited. During the week prior to admission, the patient had had an acute infection of the upper respiratory tract, paresthesias in both hands and some pain, swelling and stiffness in the joint of the right knee. Similar episodes of discomfort in the joints had been noted at intervals for several years. The patient stated that she had been a "blue baby" and had had "leakage of the heart" for many years. (There was no clinical evidence to support this.) Five years before, the uterus, both fallopian tubes and both ovaries had been removed.

During the examination the patient experienced several severe exacerbations of pain, the second of which ceased after the inhalation of amyl nitrite. The temperature was 98 F., the pulse rate 70 and the respirations 18 per minute. The blood pressure, taken in each arm, was 135 systolic and 90 diastolic. Though many teeth were missing, there were no artificial dentures. No significant cervical, pulmonary, cardiac or abdominal abnormalities were noted. The white blood cell count was within normal limits. During this examination the patient experienced a sense of a mild chill, which was not, however, followed by any elevation of temperature.

Roentgenograms of the chest (anteroposterior, oblique and lateral views) revealed no foreign body or other abnormality. She then was given a small amount of cotton to swallow. This was followed by the administration of a thin barium sulfate mixture by mouth, but no delay in the passage of the barium sulfate was evident fluoroscopically and films revealed no adherence of barium sulfate to any cotton fibers which might have been engaged by a sharp object in the esophagus. While under the fluoroscope the patient experienced no discomfort on swallowing but designated the cardiac area as the site of her pain. In the interim, she had been given ¼ grain (.015 Gm.) of morphine sulfate, with no relief of symptoms. Because of the obscurity of the clinical picture, it was decided to observe the patient for the few remaining hours of the night. Efforts meanwhile had been

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directed toward procuring the remaining contents of the plate from which she had been eating at the time the accident occurred.

On the following morning the status remained essentially unchanged except that several doses of morphine sulfate had failed to give any relief from the severe pain. The patient, however, was now aware of an increase in her pain after swallowing. No alterations in the pulmonary or cardiac findings were noted (Dr. E. M. Barton). An electrocardiogram showed no evidence of cardiac abnormality.

In view of the negative findings with respect to the chest, esophagoscopy was performed with the region under local anesthesia. The cricopharyngeal sphincter was passed with ease, and evidence of recent abrasion of the esophageal mucosa was observed on the right lateral wall, ahead of the advancing instrument. At a distance of 27 cm. from the upper alveolar margin (corresponding to the region where the left main bronchus lies in proximity to the esophagus) a large flat piece of bone was seen lying in an oblique position within the esophageal lumen. The presenting margin of the bone was smooth. This margin was grasped and rotated gently and the bone was removed without any force and without bleeding. The piece measured 22 by 20 by 2 mm. Three sides of it were smooth, while the fourth was irregular, with five or six jagged points of varying size, roughly resembling a picket fence. The patient experienced immediate relief from the severe pain in the thorax.

On her return to the ward, a relative was waiting with a large rib bone, with a defect into which the fragment of bone fitted exactly. Convalescence was uneventful, and the patient left the hospital on the second postoperative day.

COMMENT

This case is of interest for several reasons. An adult patient had no knowledge or suspicion of having swallowed a foreign body. Though it is a well known fact that persons with artificial dentures are often victims of their inability to note the presence of bones in food, they usually experience discomfort when a foreign object passes the pharynx or lodges in the esophagus. This patient did not wear artificial dentures, nor was she eating meat at the time of the accident. Her first and outstanding symptom was extreme penetrating pain in the midthoracic region, with radiation to the back. There was no dysphagia or odynphagia, although an increase in discomfort after swallowing was noted some hours later. (The circumstances were also unusual in that spareribs rarely disintegrate from boiling.)

The onset of a pneumonic process, embolism or a cardiac disturbance would have contraindicated any endoscopic procedures, and esophagoscopy was deemed inadvisable until these possibilities had been excluded. Substernal pain radiating to the back was suggestive of the presence of a foreign body in the thoracic part of the esophagus, but the absence of dysphagia and odynphagia was misleading.

The roentgenograms, examined subsequently, were still negative. In retrospect, a small capsule containing barium sulfate might well have been used to demonstrate delay in the esophagus, although cotton failed to afford evidence of the retention of barium sulfate. In the majority of instances the ingestion of cotton fiber has been found to be of value. The cotton is arrested by the foreign object, and, in turn, barium sulfate adheres to the cotton. The presence of foreign bodies, particularly bones, is thus usually satisfactorily demonstrated in roentgenograms.

122 South Michigan Avenue.

CEREBRAL AIR EMBOLISM FOLLOWING ANTRAL IRRIGATION

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Air embolism following antral irrigation is a rare but most serious complication. The average otolaryngologist irrigates many hundreds of maxillary sinuses yearly. The procedure constitutes a valuable diagnostic and therapeutic measure—simple, effective and usually harmless in the hands of a competent careful surgeon. In office routine it is the most common operation. Any avoidable complication, however rare, should be given careful consideration.

The current literature reveals little mention of air embolism following antral puncture. In 1920 Gording [1] directed attention to the subject in a rather extensive article. Recently, Blegvad,[2] in a paper on air embolism consequent to inflation of the middle ear, discussed the prevention of air embolism in cases of puncture of the maxillary sinus. Turner [3] stated:

> Alarming symptoms and even fatalities have occurred associated with exploration of this sinus. They are rare, however, and have followed, as a rule, the attempt to blow the discharge from the cavity into the nose, instead of using the douche. Possibly the explanation of the symptoms is to be found in the production of air-embolus. It is wiser to avoid insufflation of air.

Recently, Ramberg [4] has written on the risks associated with catheterization of the eustachian tube. He reported a case in which air embolism followed inflation of the tube. His patient apparently had both a cerebral and a cardiac embolism. A sudden apoplectiform seizure was accompanied by conjugate deviation of the eyes to the left, snoring respirations, dilation of the pupils, which did not react to light, red cyanosis followed by pale cyanosis, cessation of respiration and cardiac standstill. Artificial respirations restored breathing, and consciousness was regained.

Cerebral and cardiac air embolism following the introduction of air into other cavities of the body is not uncommon. It is said to occur in a mild to severe degree, once in every 1,000 pneumothorax treatments. It has followed the introduction of air into the bladder, the uterus and the knee joint and the injection of varicose veins and has occurred as a complication of labor. Guthrie [5] stated that many unexplained deaths result from air embolism, especially in operations on the neck. He reported 3 cases of serious and 1 case of fatal air

1. Gording, R.: Ann. Otol., Rhin. & Laryng. **29**:293-365 (June) 1920.

2. Blegvad, N. R.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:363-378, 1940.

3. Turner, A. L.: Diseases of the Nose, Throat, and Ear, ed. 3, Baltimore, Williams & Wilkins Company, 1932, p. 79.

4. Ramberg, J.: Acta oto-laryng. **28**:482-492, 1940.

5. Guthrie, D.: Wien. klin. Wchnschr. **50**:1667-1670 (Dec. 10) 1937; abstracted, J. A. M. A. **110**:541 (Feb. 12) 1938.

embolism, occuring during thyroidectomy. Incision into the lateral sinus is rarely followed by this complication. Blegvad [2] encountered 4 cases of air embolism following inflation of the middle ear. In 1 of these the termination was fatal.

It has been my custom and practice in clinics elsewhere to conclude the antral irrigation by insufflating air from an electric or hand pump, blowing the remaining saline solution from the antrum. This technic, I am convinced, is faulty and dangerous. The same precautions should be observed in removing air bubbles from the syringe in antral irrigation as in venipuncture. Air should not be used to blow the secretions into the nose or to determine the patency of the ostium.

The symptoms of cerebral air embolism are characteristic. The onset is abrupt, occurring immediately after the insufflation of air. The patient may complain of local pain and exhibit great anxiety and marked restlessness. Sudden pallor is followed by cyanosis, convulsive twitching, generalized convulsions, irregular pulse, loss of consciousness and irregular stertorous respirations. Death follows respiratory paralysis or cardiac standstill.

In embolism of milder degree the restlessness may amount to extreme agitation, alternating with periods of coma. Temporary hemiplegia, blindness from air in the retinal arterioles, confusion and emotional disturbances may manifest themselves.

Air embolism is frequently misdiagnosed as cocaine poisoning. Coma and sudden death following the application or injection of potent solutions of cocaine in sensitive persons have been described, and indeed the symptoms may resemble those seen in air embolism. When mild, cocaine intoxication is marked by excitement, talkativeness, acceleration of the pulse, dilatation of the pupils, a general feeling of well-being and slight convulsive movements. In the event of the absorption of large amounts of cocaine, fainting and collapse occur, with a slowing and weakening of the pulse, shallow respirations and general depression followed by death. Convulsive seizures are not common and usually are entirely absent. The factor of insufflation of air is not present.

The mortality from air embolism is said to vary from 15 to 50 per cent. A patient surviving the first ten or fifteen minutes has an excellent prognosis. Most neurologic lesions secondary to the introduction of air into the cerebral circulation are transient. The mechanics of the production of air embolism following antral irrigation is not clear. Embolism may occur at the first or any subsequent irrigation. It may follow little or considerable trauma; it has followed puncture of the inferior meatus and catheterization of the natural ostium. Sudden death results from cardiac-coronary embolism or cerebral embolism following the entrance of air under high pressure into open ulcerated or traumatized vessels. The symptoms depend on the amount and localization of air in the vascular system.

REPORT OF A CASE

The following 2 cases, in 1 of which the complication was fatal, illustrate probable cerebral air embolism following antral puncture. In the first case the Lichtwitz trocar was used by the inferior meatal

route, and in the other a curved cannula was used to catheterize the natural ostium, with an apparently mild amount of trauma.

CASE 1.—A 24 year old Negro after anesthetization with a 2 per cent solution of cocaine underwent a diagnostic puncture of the right maxillary sinus via the inferior meatus, without incident. After the insufflation of air and the withdrawal of the Lichtwitz needle the patient had a severe convulsion. The breathing became stertorous and soon ceased. Prolonged administration of artificial respiration and stimulants of all types failed to revive him.

CASE 2.—A 24 year old white woman had acute maxillary sinusitis on the right, of three weeks' duration. The middle meatus was anesthetized with a 2 per cent solution of cocaine, and a curved cannula was passed into the natural ostium apparently without incident and with only mild trauma. Pus, grade 3 plus, was irrigated from the antrum, and air was insufflated. Immediately on withdrawal of the cannula the patient had a severe generalized convulsion, with stertorous breathing. On recovery from the convulsion she remained in partial coma and exhibited extreme restlessness. Difficulty was experienced in keeping her from falling off the bed. She was removed to the hospital, where she continued to show marked agitation. There it was learned from her sister that the patient had failed to supply the information that she had been placed on a diabetic diet by another physician. In spite of the fact that the blood sugar was 312 mg. per hundred cubic centimeters the consulting physician, Dr. S. C. Hall, felt that the lack of acetone bodies in the urine and the absence of the usual picture of diabetic coma rendered the diagnosis of diabetic coma not tenable. He suggested the diagnosis of cerebral air embolism similar to that following artificial pneumothorax.

Physical examination revealed nothing abnormal except hyperactive knee reflexes and slight weakness of the left arm and hand. On the following day this weakness became more pronounced, but on the third day it improved. There was slight edema of the lids, more evident on the right side, and slight swelling of the face. The diabetes was soon controlled. Within a week, recovery was complete.

SUMMARY

The introduction of air into the human body is always fraught with danger. Extreme caution in the avoidance of trauma and the use of low pressure is imperative in those procedures in which the insufflation of air is necessary. Irrigation of the paranasal sinuses should be conducted with care, to avoid the entrance of air under pressure into the sinuses.

Clinical Notes; New Instruments and Technics

MEASUREMENT OF THE TENSION OF THE TYMPANIC MEMBRANE AND OF THE RESISTANCE OF THE EUSTACHIAN TUBE

H. A. E. VAN DISHOECK, M.D., AMSTERDAM, NETHERLANDS

The tension of the tympanic membrane and of the ossicular chain is a value which certainly deserves in the future to play a part in otology. For any deviation from the normal tension will result in a decrease of auditory acuteness, and the finding of this change of tension is of great importance in the diagnosis and in the treatment of different diseases of the tympanum. Therefore, the determination of this value by the otologist is as desirable as the determination of blood pressure by the physician. Besides, the measurement of the tension of the tympanic membrane by means of the pneumophone[1] is not difficult, it does not take much time and it is exact.

PRINCIPLE OF THE PNEUMOPHONE

A constant low tone is delivered to the ear through a rubber tube while the air pressure on the tympanic membrane is lowered from positive to negative. The tone is heard most clearly when the pressure is the same on each side, i. e., when the tension of the membrane has been reduced to the normal. If there is stenosis of the eustachian tube, a negative pressure of — 50 cm. of water is often necessary to effect this.

The 50 cycles secondary current from the transformer is conducted to the telephone, so that there arises a tone of 50 cycles per second with overtones. The back of the telephone is perforated, in order that the same pressure may be obtained on each side of the vibrating membrane. Thus, even variations of pressure up to 500 cm. of water have hardly any influence on the source of the sound. The telephone is contained in an air-tight funnel, which conducts the sound toward the ear to be examined. The pressure is obtained by means of a Politzer balloon or an ear syringe and read from a water manometer. When the tip is closed, pressure up to 100 cm. of water can be obtained. The metal tube with rubber rim, which is intended as the connection with the ear, has been made in such a way that it can easily be kept in the ear by the patient himself and does not cause stenosis of the external auditory canal.

PREVIOUS WORK

Lucae and later Gellé (1881) described the loss of hearing through pressure on the tympanic membrane. Gellé based on this his well known symptom of oto-

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From the Ear, Nose and Throat Clinic, University of Amsterdam, Dr. A. de Kleyn, director.

1. van Dishoeck, H. A. E.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:53, 1937; Nederl. tijdschr. v. geneesk. **82**:13 (Jan. 1) 1938; **83**:3654 (July 15) 1939; Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:5, 1939.

sclerosis, namely, that in ankylosis of the stapes this increase of pressure causes no loss of hearing. Griesmann (1921) [2] suggested that the degree of the loss of hearing might be a measure of the gravity of the ankylosis. These observations,

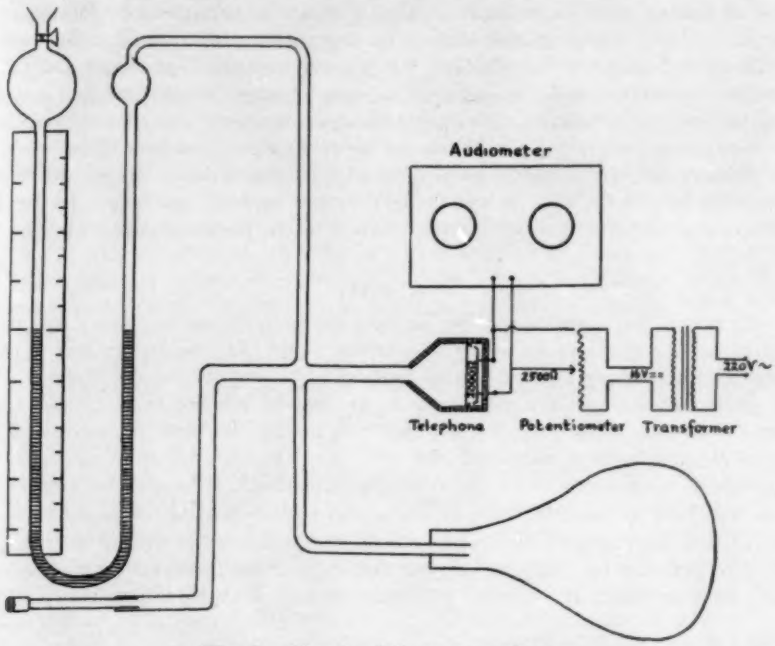

Fig. 1.—Sketch of the pneumophone.

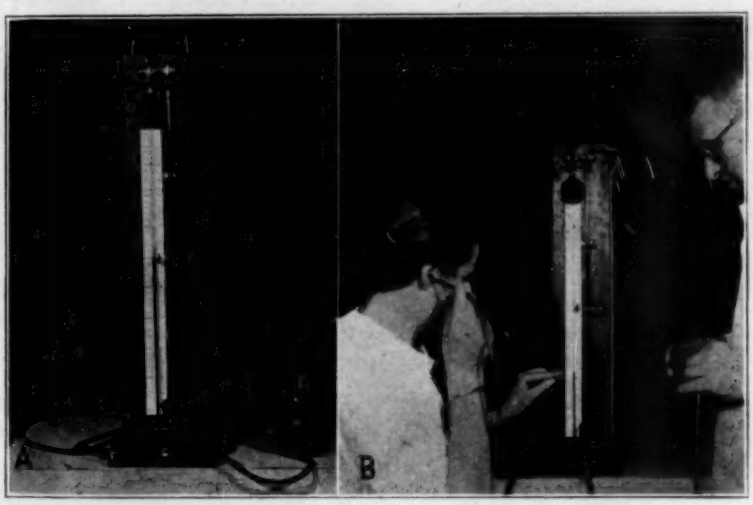

Fig. 2.—Photographs showing (*A*) the pneumophone and (*B*) the pneumophone in use. The instrument is obtainable from Lifa, Leiden, Netherlands.

2. Griesmann, B.: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **16**:47, 1921.

however, refer exclusively to positive pressure for the diagnosis of ankylosis of the stapes.

It is only in later years that some scientific studies have been published on the loss of hearing with positive and negative pressure in normal ears. Pohlman and Krans [3] (1926) measured this loss of hearing, using pressures of + 30 cm. and — 30 cm. of water. Von Békésy [4] (1932) did impedance measurements of the tympanic membrane under varied pressure, and Lüscher [5] (1938) obtained a loss of hearing by putting small weights on the umbo. However, the clinical application of the reverse principle, i. e., the use not of the depression of hearing by variation of pressure but of the improvement of hearing by correction of the tension of the tympanic membrane, as a means to indicate and measure pathologic tension has become possible only through the introduction of the pneumophone (1937).

THEORY

At first it was assumed that the pressure on the tympanic membrane was transferred by way of the stapes to the labyrinthine liquid and that the increase of intralabyrinthine pressure entailed loss of hearing. For several reasons, however, it is probable that the loss of hearing must be exclusively ascribed to an impairment of the conducting apparatus. First and foremost, the increase of tension of the tympanic membrane is responsible for this. For the vibration of every membrane exposed to air pressure from one side only is muffled, and besides, as von Békésy has indicated, at least for low pressures the loss of hearing corresponds to the increase of impedance of the tympanic membrane. But under greater negative and positive pressure the compression and extension of the ossicular chain begins to play an important part. Further particulars in regard to this will follow.

NORMAL TEST SUBJECTS

The tympanum is very sensitive to differences of pressure on either side of the tympanic membrane. In a normal ear a positive or a negative pressure of more than 2 cm. causes a decrease of hearing. So the zone of clearest hearing lies between pressures of + 2 cm. and — 2 cm. of water. This zone is easily defined, especially upward, for the positive pressure, but also downward, for the negative pressure. As the low tones exclusively are influenced by these variations of pressure, not only the strength but also the timbre of the sound changes. The tone of 50 cycles per second and the first overtones are considerably weakened, so that the high overtones become more prominent.

Variations of pressure on the tympanic membrane entail no loss of hearing in the following conditions:

1. Perforation of the tympanic membrane.
2. Adhesion of the tympanic membrane to the promontory.
3. Severe ankylosis of the stapes (Gellé).

This absence of a zone of clearest hearing can also be used diagnostically.

STENOSIS OF THE EUSTACHIAN TUBE

It has been found that the negative tension of the tympanic membrane is an accurate measure of the degree of stenosis of the eustachian tube. In the presence

3. Pohlman, A. G., and Krans, F. W.: Ann. Otol., Rhin. & Laryng. **34**:1224 (Dec.) 1925; **35**:113 (March); 632 (Sept.) 1926.

4. von Békésy, G.: Ann. d. Physik. **13**:111, 1932.

5. Lüscher, E.: Acta oto-laryng. **27**:250, 1939.

of inflammatory processes or after operations on the nasopharynx, this negative value usually varies from —10 to —20 cm. of water, according to the localization of the disease and the width of the tube. In winter a slight negative pressure (—2 to —10 cm.) is often found in entirely normal test subjects, while in summer these deviations occur principally in persons who smoke a great deal.

It appears that the symptom of an indrawn ear drum is not always accompanied by a considerable negative tension or the reverse. A slack drum with adhesion can appear indrawn without negative tension, while a resistant ear drum can have a considerable negative tension without being indrawn.

In the presence of adenoids the tension of the tympanic membrane is usually considerably lowered. Thus, it is possible, by means of a simple and quick determination, to establish with certainty presence of adenoids even in children 5 and 6 years old. An extensive examination of a great number of children is in progress.

When there is catarrh of the eustachian tube, the values range down to —50 cm., according to the gravity of the condition. After a successful air inflation the pressure is normal or positive, e. g., + 30 cm. If the positive pressure remains unchanged even after swallowing, this is a sign of a considerable degree of stenosis. The improvement of the catarrh is reflected in the lessening of the negative pressure.

MEASUREMENT OF THE RESISTANCE OF THE EUSTACHIAN TUBE

With a stationary condition almost the same negative pressure is found on repeated examination in the course of several weeks, and after air inflation the pressure, through resorption, drops again in a few hours to this level and not lower. The explanation of this fact must be that this sucking power of e. g., —30 cm. in the tympanum is sufficient in this case to conquer the resistance of the tube and to suck a little air into the ear during a deglutition. In the period between two deglutitions, the pressure again drops a little through resorption, e. g., to —31 cm. If the subject swallows now, a small air current will penetrate the tube until the pressure is again —30 cm.

So because the resistance of a tube to an air current is equal to the difference between the pressure at the entrance and that at the exit, not only the tension of the tympanic membrane but also the resistance of the tube is exactly measured by the pneumophone.[6]

Even when the tube is entirely closed, it appears that the tympanum still contains air. In such cases the pressure is not lower than about —50 cm. of water. Presumably, under this negative pressure there results an equilibrium with the blood gases, and perhaps there is some diffusion through the tympanic membrane in addition.

The determination of the resistance of the tube is of the greatest importance in the selection of pilots and caisson workers. Especially in military pilots, who must do quick ascending and diving, the permeability of the tube must be excellent. It is possible with the pneumophone to determine in a few minutes whether a pilot or a caisson worker can undergo great changes of pressure without harm.

OTHER METHODS

The methods for measuring the permeability of the tube which have been applied by Zöllner,[7] Mesrin, Kofka and others are based on the measurement of the pressure

6. van Dishoeck, H. A. E.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:248, 1939.

7. Zöllner, F.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:12, 1939.

under which air may be blown from the nasopharynx into the tympanum, either by the test of Valsalva, by catheterization or by raising the pressure during swallowing, while the movement of the tympanic membrane is observed with the ear manometer or with the ear microscope (Lüscher). But these methods have their drawbacks.

For, if it is assumed that the resistance of a tube amounts to 30 cm., this means, as I have stated already, that a negative pressure of a little over —30 cm. in the tympanum is necessary in order to make a little air go through the tube during deglutition. If an artificial overpressure of, e. g., 5 cm. is applied in the pharynx, air will also penetrate the tube during deglutition under this pressure until the negative pressure in the tympanum becomes —25 cm. But it would be quite wrong to say now that the resistance of the tube amounts to 5 cm. Besides, a comparatively great air current, i. e., a considerable overpressure, is necessary for the production of movement of the tympanic membrane.

The same objections hold good in regard to the use of the catheter, the results being further confused by leakage or pressing against the wall of the tube. With the test of Valsalva, far too high values are found, for a considerable pressure is necessary to conquer the resistance of the nasopharyngeal orifice of the tube, which resistance is absent in swallowing. As I have found from measurements, the average resistance of the orifice in normal test subjects is about 30 cm. of water. But this value is by no means constant.

TENSOR TYMPANIC MUSCLE

If the tension of the tympanic membrane is caused only by air resorption, the zone of clearest hearing immediately after air inflation is that of either positive or normal pressure, but never that of negative. Not until some hours later is the negative tension, in consequence of oxygen resorption, present again. In some patients, however, the otologist finds a positive tension after air inflation, but immediately after swallowing, this tension is again negative. In patients such as these, the lowering can have been caused only by the spasm or shortening of the tensor tympanic muscle.

OTOSCLEROSIS

For the quantitative measurements of ankylosis of the stapes, determinations of the threshold can be performed by means of an audiometer which has been connected with the pneumophone. For the tone of 512 cycles per second, the hearing threshold is determined first without pressure and afterward under a positive and a negative pressure of 50 cm. of water. In cases in which there is considerable deafness, bone conduction is to be preferred. For the bone conduction tone is also, though in a less degree, muffled by change of pressure on the tympanic membrane. In cases of complete ankylosis of the stapes, no rise of the hearing threshold is to be observed (Gellé).

RATE OF RESORPTION

When through air inflation a negative tension has been neutralized, the rate of resorption can be determined by measuring the tension every hour. An average of four hours is necessary for the pressure to drop from 0 to —20 cm. If nitrogen is used for the inflation, double that time is necessary for the lowering of the pressure.

EXTENT OF LOSS OF HEARING

The loss of hearing [8] experienced by a normal test subject in the presence of positive pressure in the external auditory canal or by a caisson worker under the

8. van Dishoeck, H. A. E.: Transactions of the Royal Netherlands Academy of Sciences, Amsterdam, 1940, vol. 43, no. 1.

conditions of his employment, corresponds with that produced by an equal negative pressure in the tympanum of a patient with stenosis of the tube or a quickly descending aviator. Likewise, the negative pressure in the external auditory canal of a test subject or in that of a caisson worker under conditions of decompression corresponds with the positive pressure in the tympanum which is produced by air inflation or by a quick ascent in an airplane.

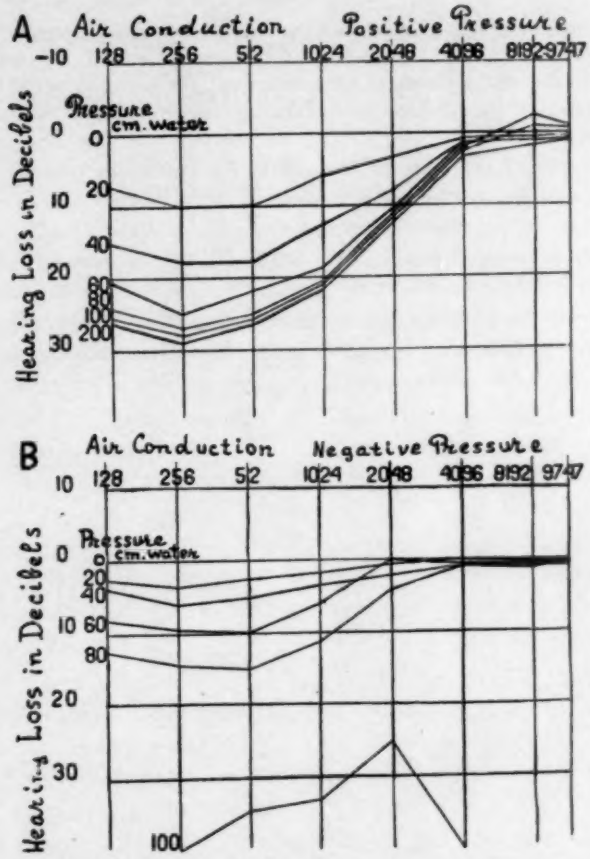

Fig. 3.—Audiograms. *A*, hearing curve resulting from increasing positive pressure. *B*, hearing curve resulting from increasing negative pressure.

The loss of hearing in the presence of positive and negative pressure is greatest for the middle tones, i. e., the area of colloquial speech. The hearing of tones higher than 2,000 cycles per second is not influenced by variations of pressure. With increasing positive pressure (fig. 3 *A*), the increase in loss of hearing becomes gradually less, and above 80 cm. there is no further increase. On the other hand, loss of hearing becomes gradually greater with increasing negative pressure (fig. 3 *B*). At a negative pressure of — 100 cm. the malleus-incus joint is probably luxated, in which event the transmission of both the low and the highest tones is much impeded.

From figure 3 *A* it appears that the loss of hearing under a pressure of + 40 cm. of water amounts to 20 decibels. If the difference between the two audiograms, before and after air inflation, of a patient with a tension of — 40 cm. is greater, or if there is a considerable deviation also in the higher tones, the possibility of complications such as swelling of the mucous membrane exudation and involvement of the labyrinth must be considered.

SUMMARY

The pneumophone serves for measuring the tension of the tympanic membrane. While the pressure in the external auditory canal is varied, a constant tone is transmitted to the ear by means of air conduction. This tone is heard most vividly when the tension of the tympanic membrane has been reduced to the normal. The pneumophone may be used:

1. To diagnose the gravity of stenosis of the eustachian tube, to control the result of air inflation and to follow the course of the disease.

2. To determine the fitness of aviators and caisson workers.

3. For the differential diagnosis of different forms of deafness, among others that associated with ankylosis of the stapes.

4. For the study of spasms and of the shortening of the tensor tympanic muscle.

5. For the measurement of the relative rate of resorption of gases in the tympanum.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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THE PARANASAL SINUSES

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(Continued from page 396)

SURGICAL THERAPY

General Considerations.—Brown [98] covers a great deal of territory in brief and simple terms, with much common sense and conservatism. He epitomizes observations after long experience, and his suggestions are worth while. He emphasizes several important rules that may well be kept in mind. 1. Carefully appraise the patient's general condition for etiologic factors. 2. Refrain from surgical measures in acute stages of sinusitis except when the severity of the condition or impending complications make intervention imperative, and then establish drainage with the least possible trauma. 3. Conserve the nasal mucosa whenever possible. 4. "Early recognition of serious complications with the proper treatment is imperative." 5. Make sure in all cases that the drainage provided is adequate.

Griffith [99] believes that in cases of chronic sinusitis in which surgical intervention is required an external approach is the better, with thorough removal of all diseased membrane. He likes the Caldwell-Luc, Ferris Smith and Sewell procedures. He also favors the use of an electric burr. He emphasizes the importance of good preoperative medication with such agents as sodium amytal, morphine and scopolamine. He recommends the use of a mucous membrane flap to line the nasofrontal duct, instead of a split graft.

Fabricant [100] presents a review of recent literature on the subject, with an attempt to evaluate the opinions of outstanding authors on the

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98. Brown, J. M.: The Treatment of Acute and Chronic Sinusitis, J. A. M. A. **115**:508 (Aug. 17) 1940.

99. Griffith, G. E.: Treatment of Chronic Sinus Disease, M. Times, New York **67**:551 (Dec.) 1939.

100. Fabricant, N. D.: The Present Status of Sinus Surgery, Internat. Abstr. Surg. **71**:105, 1940; in Surg., Gynec. & Obst., August 1940.

status of surgical treatment of the sinuses. Since it is not too lengthy and presents both sides of controversial topics, this review is easily read and informative.

Littell [101] tries to explain the reason for fear on the part of lay persons of operations on the nasal sinuses as due to misunderstanding of the role of surgical treatment and to stories of the misapplication of surgical procedure. He claims that there is no more satisfying operation than one properly indicated and skilfully executed. Success rests on proper appreciation of the influence of general constitutional states on the behavior of the nose and on adequate appraisal of the local pathologic condition. If a patient is undecided concerning an operation and a minor procedure is instituted, he should be given to understand that failure does not necessarily mean misapplication of the surgical treatment but merely that the procedure was insufficient despite the fact that in a great many similar cases it had succeeded. Patients are better prepared for more radical surgical procedures if they are told what the chances for recovery are with a lesser procedure. All this sums up to a question of the physician's knowledge of the patient's condition, local as well as constitutional, and his ability to carry out properly the best procedure for the individual case.

For diagnosis Simpson [102] places reliance on direct examination, study of roentgenograms and proof irrigation. He finds transillumination less reliable and observations made by means of the pharyngoscope often misleading. He does not think it makes much difference whether the antrum is washed through the middle meatus or punctured through the inferior meatus, as long as care is used in doing it. Fluid should never be forced through under too much pressure. It is better to wait another day for the edema to subside and the ostium to become more patent. In severe acute conditions it is better to make an opening in the inferior meatus for drainage than to do repeated painful punctures. Trauma to the inferior turbinate should be avoided as much as possible. The author prefers the Denker or the Caldwell-Luc to the Canfield operation and finds it necessary to make the flap only sufficient to cover the raw bone of the nasoantral wall. Very little packing is used. He likes the transantral approach to the ethmoid and sphenoid sinuses, which permits complete exenteration of all but the most anterior cells, which are removed through the nose. In severe acute conditions it is

101. Littell, J. J.: Nasal Surgery: The Internist and the Patient, J. Indiana M. A. **33**:412 (Aug.) 1940.

102. Simpson, W. L.: Diagnosis and Treatment of Sinusitis, Tr. Indiana Acad. Ophth. & Otolaryng. **24**: 77, 1940; Surgery of Acute Sinusitis and Its Complications, ibid. **24**:99, 1940.

better to open and drain the sinuses externally, with as little trauma as possible, than to attempt intranasal manipulation.

Sarashina [103] reviews 684 operations performed on the nose and sinuses in a three year period. In 43 of the patients otitis media developed as a complication. There were 331 radical antral operations, and 29 (8.8 per cent) of the patients had postoperative infections of the middle ear. The greatest number of these occurred in the spring.

The predisposing causes mentioned are postoperative bleeding and sore throat. A less definite cause was local reaction in the mucosa. The symptoms and prognosis were not always bad; they depended on the inciting agents and individual factors. Streptococcus haemolyticus was the organism found most frequently.

Hill [104] emphasizes the need of study of the individual case to estimate associated conditions and the probable effects on the patient. Simple measures are preferable, but if a radical operation is indicated, it should be as thorough as possible. In fulminating sinusitis the establishment of adequate drainage, with minimum work on the bone, is the objective. In cases of chronic hyperplastic sinusitis one must always consider the role of allergy.

Simpson [105] says that it is not important whether one washes the antrum through the middle or the inferior meatus. The important thing is how carefully the irrigation is done. One should never use pressure. If the solution does not come through easily, one should wait twenty-four hours and try again. Temporary edema may explain the obstruction. He sees no harm in irrigating acutely inflamed antrums if they need irrigation. Rarely does he find it necessary to remove the anterior end of the middle turbinate for drainage. Simple antrostomy may cut short an acute attack; it is done only for drainage in nervous patients and children, to permit freer irrigation without pain. In children it may be necessary only to poke a hole into the antrum with a hemostat. In orbital complications an external operation is preferable to an intranasal one. The author agrees with Mosher and Furstenburg on the treatment of spreading osteomyelitis of the frontal bone but says that osteomyelitis is not necessarily fulminating in all cases. In many the disease is localized and requires less severe measures.

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103. Sarashina, K.: Statistische Beobachtungen über die akute mittelohrentzündungen als Spätkomplikation nach den Operationen der Nase und ihrer Nebenhöhlen, Oto-rhino-laryng. **12**:939 (Nov.) 1939.

104. Hill, F. T.: Indications for Surgical Treatment in Sinusitis, Surg., Gynec. & Obst. **70**:531 (Feb.) 1940.

105. Simpson, W. L.: Surgical Treatment and Its Complications in Cases of Acute Sinusitis, Arch. Otolaryng. **32**:250 (Aug.) 1940.

Lillie, Pastore and Mousel [106] advise the making of intradermal skin tests for idiosyncrasy to cocaine, epinephrine and other anesthetics in cases in which any suspicion is aroused by the patient's history or previous experiences. They use pentobarbital sodium, morphine sulfate and atropine sulfate for premedication in practically all cases. For intranasal operations an attempt is made to anesthetize the sphenopalatine ganglion and the anterior ethmoid nerve by the direct application of cocaine on a small applicator. For operations on the sinuses injection into the second division of the distribution of the trigeminal nerve is done, either by the intraoral or by the transzygomatic route. The latter is preferred, the procedure being less painful and through a sterile field. A helpful diagram accompanies the description. For the external frontal approach, in addition to inducing intranasal local anesthesia, they block branches of the orbital nerve from the first division by injection into the skin along the supraorbital margin medial to the supraorbital foramen and along the infraorbital margin likewise. The supratrochlear and infratrochlear nerves also are blocked. A straight needle is introduced 1 cm. above the inner canthus and pushed posteriorly 2 cm., being kept in contact with the medial bony surface of the orbit. The authors try to strip the periosteum with the injected fluid. Paresis of the third cranial nerve may occur but is transient.

Korsakov [107] has performed over 300 operations on maxillary sinuses, with the patients under local conduction anesthesia. The operations were painless, and in only 12.3 per cent was there slight tenderness, due to such circumstances as difficulty in introducing the needle into the canalis infraorbitalis or into the pterygopalatine fossa or impermeability of the canal. Painful sensations were noted mostly during the manipulations in the region of the ostium maxillare. Complications were rare and of no practical importance. The use of cocaine is contraindicated. The introduction of infection was observed after injections of a solution of cocaine behind the tuber maxillare, a fact which is explained by the formation of hematoma. Injury to the arteries and local necrosis were observed only in isolated cases. There were no deaths.

Antrum.—Hennebert [108] thinks the modified Denker operation for the maxillary sinus is the most successful of all sinal procedures. He advises the widest possible resection of the anterior wall of the sinus

106. Lillie, H. I.; Pastore, P. M., and Mousel, L. H.: Methods of Producing Local Anesthesia for Tonsillectomy, Intranasal Operations and Operations on the Paranasal Sinuses, Ann. Otol., Rhin. & Laryng. **49**:38 (March) 1940.

107. Korsakov, I. V.: Clinical Observations on Conduction Anesthesia of Sphenopalatine Ganglion and Infra-Orbital Nerve in Operations on Maxillary Sinus, Zhur. ush., nos. i gorl. bolez. **17**:92, 1940.

108. Hennebert, P.: La chirurgie des sinusites, Hôpital **28**:195 (May) 1940.

and thorough taking down of the nasoantral wall posteriorly and inferiorly, but this procedure should be employed sparingly in the upward direction, so as not to damage the turbinal in any way. He believes in thorough, complete curettage of the antral mucosa and in the necessity of a good mucous membrane flap. He does not suture the wound or use packing and claims 100 per cent cures. He cannot say as much for the operations on other sinuses. The chances for success are better with operations on the posterior group of ethmoid cells than with those on the anterior group, because of more stable anatomic conditions. The author cites Mosher to the effect that when one reviews a case of infection of the ethmoid sinuses some time after the operation and sees the cavity full of recidive masses, one may well give vent to a cry of consternation, especially if one has been the surgeon. The author feels that today surgical treatment of the ethmoid sinuses is giving way to other forms of therapy, especially to displacement, which when properly and thoroughly done yields excellent results.

Richter [109] recalls Peter's [110] description (which, incidentally, is graphically illustrated in the original article), which he has found particularly useful when it is necessary to open the antrum in conjunction with the removal of premolar or molar teeth or of cysts and when doing a Caldwell-Luc operation on an edentulous jaw. The incision follows the margin of the gums at the point where they reach the crown of the tooth; the mucous membrane flap thus made offers more secure apposition, with less danger of the subsequent formation of a fistula, and also affords better access to the apexes of the teeth when these are involved simultaneously.

Eliseht [111] advocates making a bony flap of the anterior wall of the sinus in order to preserve the continuity of the infraorbital, sphenopalatine, anterior palatine and alveolar arteries and their branches and to facilitate closure without disturbing the continuity of the wall of the antrum. This is done by elevating the soft tissues and leaving the periosteum attached to the bone. The bony flap is outlined and pried upward while it is still attached to its periosteum, so that it can be replaced at the end of the operation. He claims that postoperative roentgenograms prove that the bone remains vital and unites at the margins.

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109. Richter, H.: Erfahrungen über den Zahnfleischrandschnitt nach K. Peter, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:462, 1939.

110. Peter, K.: Zur Schnittführung bei Kieferhöhlenoperationen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:340 (Aug. 9) 1930.

111. Eliseht, F. C.: Operación Caldwell-Luc: obtención de un colgajo plástico, Prensa méd. argent. **27**:1411 (July 3) 1940.

Gon [112] stresses the importance of the counteropening into the nose. He recommends making the largest possible dissection of the mucosa on the nasoantral wall and then cutting the mucosa into four flaps by two diagonal intersecting incisions. Then the four flaps are pressed into the antrum and kept in place with rubber tissue.

Matsushita and Arai [113] found that the amount of blood lost during a radical operation on the antrum varied from 25 to 430 cc., with an average of 122 cc. It was greatest in cases in which the antral disease was of a frankly suppurative form.

Frontal Sinus.—New and Dix [114] describe the procedure employed in several cases for the repair of postoperative defect of the frontal bone. Pictures and roentgenograms show excellent results. Defects or deformities due to trauma, disease or previous operation are repaired by the insertion of costal cartilage through an incision above the hair line or through an existing scar line after appropriate undermining. If more than one piece of cartilage is required, they may be sewed together with fine silk according to a previously prepared lead pattern or fixed to the frontal bone with small pieces of stainless surgical needles used as tacks.

De Sanson [115] in a rather long and detailed argument pro and con, based on his experience in over 200 operations, seeks to rationalize various procedures. He has given up the Killian method because of the bad cosmetic results. He claims to be able to prove that lack of success after operation was due to the imperfect cleaning out of an anomalous ethmoid cell. Systematic study has shown that despite the fact that a perfect technic is carried out in the same way in successive cases, the results are inconstant. This can be attributed to the irregular distribution of the ethmoid cells. Intranasal surgical procedure on the frontal sinus has only a limited field—i. e., when the frontal sinus is small and the ethmoid cells restricted or when an intranasal operation is done in association with the external operation. De Sanson's choice lies with the modification of the Jansen-Ritter operation, as described by Kos and later by Portmann. He would prevent closure of the nasofrontal duct by preserving some of the mucosa, because this prevents the

112. Gon, S.: Gegenöffnungsbildung bei Radikaloperation der Sinusitis maxillaris besonders über die Ventilmetode nach Yamamato, Oto-rhino-laryng. **13**:57 (Nov.) 1940.

113. Matsushita, E., and Arai, T.: Messergebnis der Blutungsmenge bei der Radikaloperation der chronischen Kieferhöhlenentzündung, Oto-rhino-laryng. **13**: 58 (Nov.) 1940.

114. New, G. B., and Dix, C. R.: The Repair of Defects of the Frontal Bone, Surg., Gynec. & Obst. **70**:698 (March) 1940.

115. de Sanson, P. D.: La chirurgie du sinus frontal et de ses complications, Ann. d'oto-laryng, May 1940, p. 291.

ingrowth of periosteum from the outside through the frontoethmoid suture. He avoids the use of drains, which, he says, cause irritation and contamination around the bony sutures. He also tries to preserve the middle turbinate. Of the patients whom he was able to follow up for twenty years, he found that 15 to 20 per cent required a subsequent revision. For osteomyelitis he favors the radical procedure advocated by American authors and has been able to prove regeneration of bone in 2 cases. He has not seen any results from the use of vaccines on the evolution or spread of the disease. He is convinced that the proper procedure is to attack the bone beyond the apparently diseased area, so as to avoid contamination and get all of the diseased bone out.

Anthony [116] believes that operations on the frontal sinus, are less frequent than formerly because of the use of ephedrine preparations, the discovery of allergy, early roentgen diagnosis, the treatment of antral disease and the use of sulfanilamide and its derivatives. He directs attention to the frequent mention in the literature of failure caused by blocking of the duct after surgical treatment and reviews the various methods advocated by different authors to prevent this. He quotes from Ingal's original article describing the gold tube that author used for this purpose. In 1939 Anthony saw a patient who had been operated on intranasally by Ellet in 1905 and who wore the tube for seven months. He found the duct patent, so that he could easily pass a probe into the sinus. He reports having done an external operation on 9 patients in fourteen years, using the gold tube in the newly formed duct. He removed the anterior third of the middle turbinate, the anterior ethmoid cells, ¼ to ½ inch (0.65 to 1.25 cm.) of the floor of the frontal sinus and all of the mucous membrane of the frontal sinus. He feels that the results justified the procedure, especially in those cases in which the tube was left in place for a long time. Since the tube causes no irritation and does not seem to act as a foreign body, he believes it should be used more often to insure patency of the duct, especially in large frontal sinuses, whether they are operated on by the intranasal or by the extranasal route.

Nakamura and Date [117] report 5 cases of acute frontal sinusitis. One patient was cured by a Killian operation; 3 were operated on intranasally, but in 1 of these osteomyelitis and meningitis developed; the fifth was operated on by the Killian method. Healing was delayed for three months because of the closure of the nasal passage. The authors

116. Anthony, D. H.: Use of Ingal Gold Tube in Frontal Sinus Operation, South. M. J. **33**:949 (Sept.) 1940.

117. Nakamura, F., and Date, M.: Zur Behandlung von Sinusitis frontalis acuta, Oto-rhino-laryng. **13**:56 (Nov.) 1940.

state that when the bone is involved it is not sufficient merely to open and drain the sinus. The diseased bone must be removed, and therefore the Killian operation is not sufficient. [Not having read the original, I find it hard to say what type of case Nakamura and Date were handling. It appears, however, that they are referring to cases in which the disease was fulminating. S. S.]

Ision Ponte [118] reports a case in which an operation by the Portmann technic was performed on a patient who had a positive Wassermann reaction. The condition was acute and failed to yield to palliative treatment. There was evidence of involvement of the bone. The operation, which was performed about a month after the onset, consisted of an intranasal ethmoidectomy and an external procedure on the frontal sinus. The pictures show a fairly good cosmetic result, although a depression is visible near the inner end of the brow. The author believes the frontal sinus should be considered in connection with the ethmoid as similar to the mastoid sinus, and he therefore dressed the wound open with gauze packing.

Matis [119] presents the details of his subperiosteal approach to the frontal sinus. It is the same procedure previously described by him in various foreign journals, including the *Journal of Laryngology and Otology.*

Ethmoid, Frontoethmoid and Sphenoid Region.—Morrison [120] gives the details of the external operative procedure, including preoperative and postoperative care. He advises electrocoagulation of small vessels for hemostasis unless the patient is under anesthesia induced with ether. He claims that this provides a bloodless field. He follows the technic of Ferris Smith in the main. He prefers an epidermal graft, held in place by an inflated balloon, for maintaining the patency of the frontal duct. He calls the procedure described the "Jansen-Lynch technic." A number of photographs accompany the article, showing the successive steps. As positive indications for the operation he gives the following: 1. An orbital abscess from rupture of a focus of sinal infection. 2. A chronic suppurating fistula leading into the orbit following acute orbital abscess of sinal origin. 3. A foreign body in the interior of any of the upper sinuses. 4. Mucocele, cyst and other growths. 5. Fracture of the sinal walls, with secondary infection. 6. Severe chronic infection of the sinuses, especially if there are marked local and focal symptoms. 7. Suppuration which intranasal operation has failed to cure.

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118. Ision Ponte, A.: Sinusectomia frontal, Rev. flum. de med. **4**:91 (Feb.) 1939.

119. Matis, E. I.: Die subperiostale Resektion des Processus frontalis maxillae als Zugangsoperation, Acta oto-laryng. **28**:153, 1940.

120. Morrison, W.: The Technic of the Modern External Fronto-Ethmo-Sphenoid Sinus Operation, New York State J. Med. **40**:1145 (Aug. 1) 1940.

The relative indications are: 1. Less severe chronic disease which may be amenable to intranasal operation. An external operation is more certain. 2. Continued suppuration of certain cells which intranasal surgical procedure has failed to reach.

Difficulties of the external operation are: 1. That of complete removal of all sinal mucosa from the periphery of a shallow but high and wide sinus. 2. Difficulty of reaching the sphenoid cells when there are perisphenoid ethmoid cells. Morrison describes ways of overcoming these difficulties.

The dangers of the operation are: 1. Accidental perforation of the posterior wall of the frontal or of the roof of the ethmoid sinus. 2. Trauma to the orbital contents. 3. Hematoma of the orbit due to pricking of an orbital vessel by the injecting needle or incomplete ligation of the major vessel. 4. Damage to the pulley of the superior oblique muscle. 5. Damage to the cornea through pressure. 6. Stitch abscess. 7. Postoperative hemorrhage from the nose.

Blomfield [121] is satisfied with the external operation as described by Sewell and Ferris-Smith and has done it a number of times. He has encountered cases in which intranasal operation was shown by subsequent external operation to have been incomplete. However, he has encountered 2 cases in which there was a recurrence of polyps. In one the middle turbinate had been left in place, and in the other it had been removed. In the latter case there was a continuous secretion of pus despite thorough surgical procedure. Roentgen treatment was being tried for this condition. The author thinks that the cosmetic results of this operation are not always the best and has seen some deformities that were objectionable. On the whole he is satisfied that the operation is the best so far devised. He also has had good results from the transantral approach when a radical operation on the antrum was required. However, despite control by frequent observation through the nose during the operation he feels that even this approach cannot give a sufficiently good view of the area to make possible the exenteration of the most anterior cells, particularly those of the frontoethmoid region.

McKenzie [122] presents a well illustrated article describing the transantral approach with intranasal exenteration of the ethmoid and sphenoid sinuses. He uses combined local and general anesthesia, induced with morphine and atropine and with gas and ether. The ether is given through a Branower breathing tube. He makes injections into both sphenopalatine ganglions, also the septal mucosa and the turbinates.

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121. Blomfield, B. B.: The Operative Treatment of Ethmoiditis, M. J. Australia **2**:150 (Aug. 17) 1940.

122. McKenzie, W. R.: The Diagnosis and Surgical Management of Chronic Sinusitis, Surg., Gynec. & Obst. **70**:523 (Feb., no. 2 A) 1940.

This reduces bleeding and shock. He also injects both canine fossae. If the septum is deviated, he resects it. He displaces the middle turbinate medially. If it is enlarged, he resects it by splitting it lengthwise. The ethmoid cells are opened with a Mosher curet and packed with gauze impregnated with a solution of epinephrine hydrochloride. The antrum is entered through the canine fossa, a triangular incision being made. The entire mucosa is removed. The bony nasoantral wall is then taken down, the nasal mucosa being preserved intact. The ethmoid cells are entered at the posterior-superior angle of the antral wall and the opening enlarged with a biting forceps. The gauze sponge previously placed in the ethmoid sinus is now drawn out, and all the cells below and external to the displaced middle turbinate are removed. This brings the anterior wall of the sphenoid sinus into view. This sinus is entered with a curet, and the entire anterior wall is taken down. A large window is made in the inferior meatus, a mucous flap being used to cover the bone near the floor. For packing the author uses gauze treated with nupercaine ointment. This is removed in twenty-four hours, and the sinus is irrigated after forty-eight hours.

Kirkland [123] comments favorably on the external operation but is not entirely satisfied with the cosmetic results. In 2 cases there was a tendency for polyps to recur. In one of them the middle turbinate was left in place, and in the other it was amputated for the most part. In the latter case the wound is still suppurating, and the author is trying roentgen therapy. In a case in which a transantral ethmoid operation had been done some time previously, he was surprised to find how many anterior ethmoid cells had not been reached.

Mangabeira-Albernaz [124] reviews the history of various transantral approaches to the ethmoid cells which have been used since the time of Jansen (1894) and gives the comments of authorities on their value. He is enthusiastic over de Lima's technic, which he claims is the best. He classifies the various procedures according to the tissues resected and places the de Lima technic in the group of those in which the nasal wall is resected in its superior-posterior angle and in the region of the inferior meatus. He calls the procedure a "submucous resection of the ethmoid," i. e., the resection is done under the nasal mucosa. The advantages are less bleeding, less crusting and the preservation of healthy nasal mucosa. The procedure is anatomically sound, because the ethmoid labyrinth is the point of convergence of all the sinuses. The technic is described, with illustrations. 1. The usual Caldwell-Luc approach is

123. Kirkland, H.: The External Fronto-Ethmoid-Sphenoid Approach, M. J. Australia **2**:149 (Aug. 17) 1940.

124. Mangabeira-Albernaz, P.: L'opération d'Ermiro de Lima (ethmoïdectomie transmaxillaire sous-muqueuse), Pract. oto-rhino-laryng. **2**:352, 1939.

employed, with complete removal of the anterior wall of the maxillary sinus. Care is taken to avoid the infraorbital foramen. 2. The ostium is located; if the wall arches inward, careful resection is done without injury to the nasal mucosa. 3. The ethmoid labyrinth is opened in the posterior-superior internal angle with a Struycken forceps held parallel to the nasal wall. By means of a Gruenwald or a Citelli forceps the nasal wall anterior to the ostium, i. e., the anterior-superior internal angle, is removed. This opens the ethmoid labyrinth in the region of the agger cells. 4. This opening is carefully enlarged with the principal aim of exposing the dihedral angle formed by the superior, or orbital, wall of the sinus with the lamina papyracea, or the external limit of the ethmoid sinus. All the cells can then be cleaned out. 5. The roof of the ethmoid sinus is explored. The bone is thin but resistant. Nevertheless, care must be taken. 6. The angle formed by the roof of the ethmoid sinus with the lamina papyracea is located. One should look for its posterior extremity and perforate it carefully with a curet in a posterior direction. Previously made roentgenograms will help to show the direction to the sphenoid sinus. 7. One should look for the anterior extremity of the angle between the roof and the interior wall of the ethmoid sinus and perforate it; this provides entrance into the frontal sinus. 8. The inferior meatus is opened at its middle for irrigation and drainage. No tampon, no suture and no postoperative irrigation are used. Only two dangers or difficulties may arise: (1) injury to a branch of the sphenopalatine artery and (2) penetration of the cranial cavity through the roof of the ethmoid sinus. The latter can be avoided by following the landmarks and studying the roentgenograms. The author mentions several recorded cases in which the cranial cavity was penetrated. In such cases the perforation was enlarged and careful asepsis preserved. No deaths occurred.

Nishihata [125] speaks against incomplete operations on the ethmoid labyrinth and in behalf of complete exenteration. The ethmoid cells are involved in 80 to 96 per cent of all cases of sinal empyema. His reason for advocating a radical procedure is that he has encountered many cases in which obscure nervous conditions were completely cleared up by the operation. He seems to think that these are of the category termed "ethmoiditis superficialis" by Zuckerkandl. These are the conditions which are often called "latent," because of the paucity of symptoms.

Mithoefer [126] speaks of two types of ethmoiditis—the suppurative, in which the mucosa is red, swollen and firmly attached, and the serous,

125. Nishihata, T.: Ueber die Operationstechnik bei Nasennebenhöhlenentzündungen, Oto-rhino-laryng. **13**:36 (Aug.) 1940.

126. Mithoefer, W.: How and When to Operate upon the Ethmoid Sinus, Surg., Gynec. & Obst. **70**:539 (Feb., no. 2 A) 1940.

in which the mucosa is pale, edematous and without proliferation of connective tissue except in the very late stages. Headache is not always an indication of disease of the ethmoid sinus, since it may be due to the pressure of a swollen turbinate on branches of the anterior ethmoidal nerve. For this condition he recommends infraction of the turbinate and the application of trichloroacetic acid. He directs attention to the importance of careful scrutiny of the ethmosphenoidal recesses, where one frequently finds evidences of disease of the posterior cells, such as edema, plugs of mucus and other evidence of secretion. When found in conjunction with anosmia and stuffiness of the nose, these symptoms are very significant. When the ethmoid sinus is operated on in connection with the surgical treatment of other sinuses or in the presence of extensive polyposis, the transantral approach, he states, affords the best opportunity of reaching the cells that arch above the orbital roof or extend externally into the posteroexternal angle. In the external operation, he advises extensive resection of the frontal process of the superior maxilla and less radical removal of the lamina papyracea. He deems it advisable also to spare the mucosa of the posterior aspect of the nasofrontal duct.

After endonasal or transantral operation, Findlay [127] advises against packing except in the presence of severe bleeding. Faced with this, he uses a solution of acriflavine (1:1,000), a solution of mercurochrome (1:1,000) or paraffin-treated gauze for twelve hours. Subsequently he employs frequent applications of mild protein silver or of 10 per cent silver nitrate after proper shrinking and suction. For recurrent polyposis after operation, if there is no suppuration, he finds radium or high voltage roentgen rays efficacious. He advises using the therapeutic agent a few days after the operation has been done and quotes both American and English authors as to dosage and technic. He criticizes other authors for failing to state the roentgen dosage and points out the danger of septal necrosis if this is uncontrolled. He advises the use of a 25 mg. of radium element in 2 mm. of platinum. This is covered with rubber and placed in the ethmoid cavity, on each side. It is left in place for eight hours. This gives 200 milligram hours to each side, or 3,800 r units at the surface and 1,050 r at 1 cm. in plane. "If the resulting erythema in the area irradiated is not pronounced, a further dose may be given in a month's time, half the original dose." If roentgen rays are used, he advises administration of a ⅔ erythema dose or a full radiation dose amounting to 2,400 r, i. e., 1,600 r to the area, with 4 mm. of copper and aluminum. This dose is divided and given in six to eight sittings.

127. Findlay, J. P.: The Treatment of Ethmoid Suppuration and Polyposis After Operation, M. J. Australia **2**:151 (Aug. 17) 1940.

NONSURGICAL THERAPY

General Considerations.—Williams and Mousel [128] present an excellent general summary of sinusitis. They state that the conditions usually necessary to cause sinusitis are:

> (1) Interference with the vasomotor control mechanism that leads to anemia of the membranes and changes in the mucous blanket; (2) interference with ciliary movement by bacterial toxins, drugs or drying; (3) mechanical interference with the pathways of discharge due to abnormal structure and (4) unusual stress placed on the defense mechanism by unfavorable environmental conditions.

In discussing chronic sinusitis they direct attention to the infrequency of local symptoms. General symptoms predominate, such as a run-down condition, ready fatigue, frequent respiratory infections or chronic disorders of the lower respiratory tract. The details of diagnostic examination are gone into carefully. Williams and Mousel note the difficulties in eliminating the allergic element in many cases in which specific allergens are not discovered. They are encouraged by the good results obtained from the administration of histaminase (2 to 4 tablets before each meal) in such cases. They also use pack treatments and suction. The importance of diet is stressed. Roentgen treatment and physical therapy have been found to be less efficient. The authors decry prolonged and frequent irrigations of the antrum.

Dubail [129] presents a brief summary of the therapy. He distinguishes acute maxillary conditions of rhinogenous origin from those due to dental infection. His therapeutic hints offer nothing new and follow pretty generally the accepted practices. In discussing the Proetz displacement therapy, he claims that in many instances objections to this procedure arise from ignorance of the proper method of carrying it out. He claims that this is the method of choice in the treatment of disease of the ethmoid and sphenoid sinuses.

Briant [130] does not believe that the short wave has any greater value in the treatment of acute sinusitis than any other heat-producing agent, particularly the infra-red lamp. He believes that careful irrigation through the natural ostium can be done in 80 per cent of the cases in which the disease is acute and that it is valuable for the relief of pain in that it removes secretions which are not draining well spontaneously. Other methods commonly in vogue are discussed and approved.

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128. Williams, H. L., and Mousel, L. H.: Diagnosis and Treatment of Chronic Disease of the Paranasal Sinuses, Ann. Otol., Rhin. & Laryng. **49**:466 (June) 1940.

129. Dubail: Le traitement médical des sinusites, Hôpital **28**:55 (Feb.) 1940.

130. Briant, T. E.: Problems in the Conservative Treatment of Sinusitis, Canad. M. A. J. **42**:260 (March) 1940.

Orton [131] discusses the rational treatment for sinusitis with regard to the natural reparative processes and advises cooperation with the allergist. Sulfanilamide should not be used indiscriminately.

Irrigation.—Krepuska [132] describes a trocar and cannula by which it is possible to puncture and irrigate the antrum and have the return flow unobstructed. This device is similar in structure to the Krause instrument but has an offshoot, through which the fluid returns. After the antrum has been entered, the trocar is withdrawn and a small metal tube is passed through the cannula, through which the irrigating fluid is instilled. This device is of special value when the natural ostium is obstructed, since it permits free egress of thick secretion without undue pressure.

Bowers [133] cites his record of 2,580 irrigations through the middle meatus with only 6 mishaps. A blunt cannula was used. He found 28 per cent of the openings tight and 65 per cent large. In 6 per cent of the cases the size of the opening was not noted, and in 1 per cent the opening was very tight. As to position, in 33 per cent of the cases he found the antral opening large and back; in 32.5 per cent, large and forward; in 12 per cent, tight and forward; in 11 per cent, tight and back; in 8 per cent, high, and in 3 per cent low. He considers the procedure less traumatic than puncture and better tolerated by most patients, producing less hemorrhage and less pain and shock. He finds that a deep middle meatus usually means a small antrum close to the orbit. The mishaps noted were penetration of the orbit (5 cases) and penetration of the cheek (1 case). There were no bad after-effects.

Kanath [134] tells why certain measures, such as irrigation, the application of strong antiseptics and insufflation, formerly widely employed, are not regarded as safe in the light of the new knowledge of the physiology of the nose. He recommends the use of ephedrine in physiologic solution of sodium chloride, with the patient in the Parkinson position. This is followed by an attempt at displacement with the same solution, the patient still in the Parkinson position. The displacement is accomplished by voluntary negative pressure induced by forcible inhalation with the nasal orifices closed and the mouth closed. [This procedure has been described several times in the South American literature. S. S.]

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131. Orton, H. B.: Non-Surgical Therapy in Acute Sinus Disease, Surg., Gynec. & Obst. **70**:534 (Feb., no. 2 A) 1940.

132. Krepuska, S.: Ein neues Instrument zur Kieferhöhlenpunktion, Monatschr. f. Ohrenh. **74**:128 (March) 1940.

133. Bowers, W. C.: Lavage of the Antrum with a Blunt Instrument Through the Middle Meatus, Ann. Otol., Rhin. & Laryng. **49**:1037 (Dec.) 1940.

134. Kanath, V. R.: Rational Intranasal Medication, Antiseptic **37**:377 (May) 1940.

Parkinson [135] recommends the following formula as fulfilling the requirements of a physiologic solution containing ephedrine:

1. Water + ephedrine sulfate to 1 per cent + sodium chloride to 0.68 per cent.
2. Boil.
3. Keep in boiled screw-capped bottles.

He recommends the use of tap water when this is naturally alkaline; if distilled water is used, it is necessary to alkalinize it, to bring it up to the physiologic p_H, 7.6. The advantages claimed for the solution are (1) that the values for osmotic pressure and hydrogen ion concentration are physiologic, (2) that it is sterile, yet contains no antiseptic, (3) that it is stable in physical characteristics and effectiveness and (4) that it is odorless, colorless and clear.

Tremble [136] describes a trocar which combines the features of that described by Laurens and that described by Lobell, by means of which it is possible to puncture the anterior wall of the sphenoid sinus safely even though this may have to be done blindly in many cases. The trocar has marks to indicate the depth to which it can be inserted safely, and the terminal portion is bent downward so as to avoid penetrating the posterior wall of the sinus. Tremble also describes a method of visualizing the cribriform plate in this procedure by means of a strip of adhesive tape placed along the side of the temporal area to parallel Reid's base line, which is a good guide to the location of the cribriform plate. He uses on the patient a pair of spectacles each of which has a black line drawn across the plain glass at the level of the inner canthus, to indicate the level of the cribriform plate.

Gundrum [137] presents a summary of his results in the treatment of subacute and chronic sinusitis. The percentage of good results is as follows: from foreign proteins, 25; from ephedrine and neo-synephrine, 58; from bacterial antigens, 71. Rechecking 100 cases selected at random after two to six years, he found that 31 patients had recurrences or reinfections after mild attacks of acute rhinitis; 24 returned for treatment, and the condition yielded to fewer treatments than in the first instance; 68 had no recurrence.

Drugs.—Walsh [138] found the subcutaneous use of vaccine disappointing in his own experience and in the controlled investigations recorded

135. Parkinson, S. N.: Preparation of a Physiologic Solution for Nasosinal Treatment, Arch. Otolaryng. **32**:959 (Nov.) 1940.

136. Tremble, G. E.: Irrigation of the Sphenoid Sinus, Arch. Otolaryng. **32**:952 (Nov.) 1940.

137. Gundrum, L. K.: Treatment of Sinusitis by the Displacement Method: Report of Eight Hundred Cases Using Ephedrin, Neosynephrin, Bacterial Antigens and Foreign Proteins, Laryngoscope **50**:989 (Oct.) 1940.

138. Walsh, T. E.: Intranasal Vaccine Spray: Its Use in Prophylaxis Against the Common Cold, Ann. Otol., Rhin. & Laryng. **49**:875 (Dec.) 1940.

by others. He reports results from the use of a special vaccine applied locally in the nose, as a spray. Cultures of nasal material taken from patients with acute and chronic respiratory infections were grown on agar, and typical colonies were identified and grown separately on appropriate mediums. After proper killing of the bacteria, centrifuging and washing, suspensions were made in the following proportions:

	Parts
Staphylococcus albus, hemolytic and nonhemolytic, of each	1
Staphylococcus aureus, hemolytic	1
Streptococcus, alpha, beta and gamma, of each	2
Pneumococcus, all types, of each	1
Diphtheroid bacilli	1
Friedländer's bacillus	2
Bacillus influenzae	2

The solution was diluted so that the final suspension was approximately equal to that of 1,000,000,000 staphylococci per cubic centimeter. Merthiolate (1:10,000) was added as a preservative. Walsh recommends a De Vilbiss no. 28 atomizer because it is small and is easily cleaned when not in use. Six puffs of the vaccine are snuffed into the nostrils before the patient retires, each night for three weeks and again, after one week's rest, for two weeks. The applications are continued for alternate two week periods during the "cold year," from September to May. Only patients who had no chronic sinal disease or faucial infection were used for the experiment. Those who were used as controls were treated with a solution of similar appearance. Neither the patient nor the physician knew which was the vaccine and which the control solution. The results were as follows:

		Per Cent
Vaccine, 74 patients:	Good results	62
	Fair results	15
	Bad results	23
Control solution, 74 patients:	Good results	50
	Fair results	None
	Bad results	50

The chief difference was in the number who claimed fair results. This meant that they had fewer colds than they had during the previous season or less severe attacks.

Bowers [139] holds that the antrum is the keystone of most sinal disease. A review of 1,000 private cases showed that in 96 per cent there was involvement of the antrum, either alone or in conjunction with

139. Bowers, W. C.: The Treatment of Sinusitis, West Virginia M. J. **36**: 489 (Nov.) 1940.

the other sinuses. When the ethmoid cells alone are affected, he believes that in most cases the condition is due to allergy. He mentions that the continued use of mild protein silver tends to produce argyrosis and says that since its effect in packs is due to the osmosis it induces, the same result can be obtained without danger. He likes ephedrine in physiologic solution of sodium chloride for shrinking and decries the use of oily solutions except for chronic crusting. He has had good results from chemotherapy in severe forms of sinal disease and has not seen any late bad results due to masking, such as those reported in connection with mastoiditis. He believes that the best way to avoid masking is to follow the case with frequent roentgenograms, which will show osseous involvement. As a rule, if there is no result after seventy-two hours of chemotherapy, he thinks that other measures are indicated. Failure to respond to chemotherapy may be due to the absence of antibodies, in which case the use of serums, vaccines and immunotransfusions is indicated.

Ayuyao, Yambao and Simuangco[140] experimented with a series of 224 patients who had subacute and chronic antral suppuration. The patients were divided into groups and treated by irrigation with various solutions, to determine, if possible, the effectiveness of each solution. The following solutions were used: (1) physiologic solution of sodium chloride; (2) a 1:10,000 solution of potassium permanganate; (3) physiologic solution of sodium chloride followed by the instillation of 3 to 5 cc. of a 20 per cent solution of a colloidal silver preparation (collargol) or of the same amount of a 2.5 per cent solution of azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate); (4) a 1:10,000 solution of potassium permanganate followed with physiologic solution of sodium chloride and then with a 20 per cent solution of a colloidal silver preparation (collargol) or with a 2.5 per cent solution of azosulfamide. The authors found that the highest percentage of recoveries followed the use of the solution of azosulfamide after irrigation with a saline solution. They note that the solution of azosulfamide was less effective in cases in which the discharge was mucoid than in those in which it was frankly purulent.

Young and Charteris[141] recommend the packing of gauze soaked in a 1 per cent proflavine oleate into a sinal cavity, to combat foul odor and postoperative sloughing, especially in cases of cancer in which radium has been used and in cases of localized osteomyelitis. This controls the odor and sepsis, the patients having less discomfort and

140. Ayuyao, C. D.; Yambao, C. V., and Simuangco, P.: A Comparative Study of Some Treatments of Inflammation of the Maxillary Antrum, J. Philippine M. A. **20**:451 (Aug.) 1940.

141. Young, G., and Charteris, A. A.: The Use of Proflavine and Acriflavine in a Throat and Nose Department, Brit. M. J. **2**:489 (Oct. 12) 1940.

fever than otherwise, and keeps the cavity clean. In their wards Young and Charteris also use a 1:5,000 solution of acriflavine as a gargle, to prevent scarlet fever. They find this especially useful in postoperative care and have noted a marked reduction in the number of cases complicated by scarlet fever with the routine use of this procedure.

Szilagyi [142] recommends a modified treatment for a subacute purulent process of the maxillary cavity, excluding conditions with origin from other purulent foci. His treatment consists of slow irrigation of the cavity with a 1 per cent hydrogen peroxide solution followed by injection into the cavity of 20 cc. of a 5 per cent acriflavine hydrochloride solution. He claims that he has been able to control the pathologic process by this treatment in almost every case. Patients whose condition has been neglected, if unimproved after four to five treatments should be operated on radically. He explains further that there is no danger of embolism from the formation of gas that accompanies the use of solutions of hydrogen peroxide.

Roentgen Rays and Radium—There is still considerable difference of opinion as to the value of roentgen therapy in sinusitis, due no doubt to differences in criteria regarding pathologic conditions and to varying standards of appraisal in the post-treatment periods. On the whole, one will find that the roentgenologist is more enthusiastic than the rhinologist, and yet reports of carefully controlled studies prove that radiation therapy has definite merit in certain restricted types of disease.

Maxfield and Martin [143] review numerous reports from the literature on the value of roentgen therapy in cases of sinal disease and find considerable variation in the percentages of cure or improvement reported. Several cases observed by them are cited, to show that this treatment can achieve excellent results; roentgenograms of patients before and after treatment are shown. In most of these cases the disease was chronic. The authors express the belief that when palliative treatment has failed, one should try roentgen therapy before surgical intervention, since the former has been proved beneficial in cases of simple hyperplasia. They agree with Butler and Wooley that a single full dose is more beneficial than multiple fractioned doses. They point out that the treatment is contraindicated if there is inadequate drainage because of obstruction in or about the ostiums and also when the presence of a tumor is suspected. They also insist that patients treated with roentgen rays should be followed up closely by a rhinologist, for proper evaluation.

142. Szilagyi, L.: New Treatment of Sinus Infection, Orvosi hetil. **84**:370 (July 20) 1940.

143. Maxfield, J. R., and Martin, C. L.: Evaluation of Roentgen Therapy in Sinus Disease, Radiology **34**:300 (March) 1940.

As an experiment, to evaluate the effect of roentgen therapy in acute sinusitis, Williams and Popp [144] treated 56 patients. These were divided into two groups—those in whom the involvement was moderate and those in whom it was severe. The patients in whom involvement was considered moderate were those who could tolerate repeated pressure over the sinus and who did not experience pain severe enough to interfere with sleep. The patients in whom involvement was regarded as severe were those who resisted repeated pressure over the sinus or who required morphine for sleep. Most of the patients also received Dowling packs, although this treatment alone had failed to give relief to some. Others received hot fomentations, and some who were severely ill received sulfanilamide. In 18 cases in which the disease was severe, the symptoms had been present more than five days. Nine patients had relief after the first treatment, 2 after the second treatment and 1 after the third treatment. Six obtained no relief. (One of these had an abscess in the frontal lobe of the brain.) In 25 cases in which the disease was moderate, pain and other symptoms had been present less than five days; 8 patients had relief after the first treatment and 1 after the second treatment; there were no patients who failed to obtain relief. Sixteen patients had pain and other signs more than five days; 8 obtained relief in six hours after the first treatment and 6 experienced moderately good results. The dose used was as follows: for patients with symptoms of less than five days' duration, 50 r.; for those with symptoms of more than five days' duration, 75 to 100 r. The authors were impressed by the striking relief from pain, headache and other manifestations in most cases. They believe the treatment to be a valuable adjunct for symptomatic relief.

Gertzenstein [145] reports good results in 8 cases, in only 1 of which the condition was acute. Six patients with chronic involvement, who had pain and discharge, were relieved, as was 1 who still had pain after operation. The one with involvement received 150 r. Those with chronic involvement received three treatments each, at twenty to thirty day intervals, the total dose being 360 to 400 r. The author advocates the use of roentgen rays early for acute conditions but states that the usual procedures should be employed also.

Gatewood [146] found that a check-up of 22 patients treated with roentgen rays yielded unfavorable conclusions. Four patients showed

144. Williams, H. L., and Popp, W. C.: Roentgen Therapy for Acute Sinusitis, Ann. Otol., Rhin. & Laryng. **49**:749 (Sept.) 1940.

145. Gertzenstein, S.: Radioterapía de los procesos inflamatorios agudos; radioterapía de las sinusitis maxilares, Semana méd. **47**:1254 (May 23) 1940.

146. Gatewood, E. T.: Of What Value Is Roentgen Therapy for Sinusitis? Arch. Otolaryng. **31**:275 (Feb.) 1940.

relief clinically, and this was corroborated by roentgenograms. Eight were symptomatically improved, but roentgenograms showed only moderate, not complete, recession of the membranes. None of these, however, was clinically normal; 3 of the 8 showed purulent discharge on antral irrigation. Ten patients were not improved clinically, and roentgenograms showed no improvement. Four of these were operated on by the Caldwell-Luc technic. The mucosa was found to be hyperplastic and polypoid. Streptococcus was present in the secretions. Sections made in these cases showed no evidence of changes that might be due to the roentgen rays. The author believes that the checking up of patients treated with roentgen rays should include a careful taking of the history and a complete examination, since frequently roentgenograms do not indicate the true condition.

Of 25 cases in which there was chronic involvement of up to ten years' duration and in which many treatments, including radical operation in some cases, had been given previously, Molinari and Lemas Ibañez [147] obtained cures in 50 per cent and great improvement in 40 per cent through roentgen therapy. They are impressed with the beneficial results of this therapy in acute involvement when it is given in smaller doses, provided drainage has been effected. Their average dose for patients with chronic involvement was about 400 to 700 r; in exceptional cases they have given up to 1,000 r.

Cherubino and Toniolo [148] report their results in 13 cases of acute, 5 of subacute and 20 of chronic involvement, with doses totaling from 250 to 1,200 r, depending on the case. The distance was 30 cm., with 3 mm. of aluminum filter, and the current used was 140 kilovolts and 2 milliamperes. All the acute as well as the subacute infections were cured, and of the patients with chronic involvement 12 were cured and 5 improved, while the condition of 4 was doubtful. Several of the patients with chronic involvement also were operated on in addition to being given both medical and roentgen therapy.

Firor and Waters [149] found that 34 per cent of their patients were cured by roentgen therapy; i. e., all symptoms were relieved and the sinuses appeared clear on roentgen examination. The condition of 38 per cent was markedly improved; i. e., the symptoms were completely or almost completely relieved, the sinus remaining slightly cloudy on

147. Molinari, J. L., and Lemas Ibañez, A.: Roentgenterapía de las sinusitis, Día méd. **12**:904 (Sept. 30) 1940.

148. Cherubino, M., and Toniolo, G.: Sulla roentgenterapia delle sinusiti fronto-etmoido-masellari acute e croniche, Boll. d. mal. d. orecchio, d. gola, d. naso **58**:171 (May) 1940.

149. Firor, W. B., and Waters, C. A.: Present Status of Roentgen Treatment of Sinus Infection, Am. J. Roentgenol. **42**:857 (Dec.) 1939.

roentgen examination. They cite other authors, whose work has been abstracted previously in these reviews, and conclude that roentgen therapy is useful in cases in which there is subacute or chronic involvement, when other therapy short of surgical intervention has failed and when polyps are not present. The best results were in cases in which lymphocytic infiltration predominated.

For recurrent polyposis of the ethmoid sinus Robinson [150] advises the use of radium, a total of 600 to 800 milligram hours in three to four divided doses administered at ten day intervals. The capsule is screened with 0.5 mm. of platinum applied intranasally in the ethmoid region. Radium induces fibrosis of the subepithelial tissues. Good results are reported, but no statistics are given.

Short Wave.—Dalmark and Goldin [151] find that in their cases short wave therapy has not achieved the results that are claimed by others. A series of 13 patients with chronic antral disease were studied. Each received from 13 to 30 treatments, lasting twenty minutes each. Twelve patients had been treated previously by puncture irrigation; in 12 the abnormal condition was demonstrated by roentgen rays and in 8 by contrast mediums as well. Eleven patients were examined roentgenologically after the treatments. Twelve showed no objective signs of improvement. In 4 some improvement was noted subjectively.

Paterson [152] is convinced that the short wave increases circulation and dilates capillaries but finds no evidence of an increase in nasal temperature. Treatment by this means is contraindicated for patients with cardiac disease, tuberculosis and carcinoma. One must remove obstructions before using it. The results are not good in allergic patients or in those with polyps. As a rule, the results are good in children. In the latter the treatment is preceded by shrinking of the passages and by the removal of pus and mucus from the nasal chambers. The average treatment takes fifteen to twenty minutes. Six to eight treatments are necessary for acute sinusitis. Paterson uses a 6 meter wavelength and gives only enough heat to make the patient comfortably warm—not hot. In a series of 126 patients with acute and subacute involvement he found 96 with improvement; in 22 the results of the treatment were questionable, and 8 experienced no improvement. Since using this treatment he has had to do fewer irrigations than formerly. [A question arises whether this proportion with improvement does not

150. Robinson, G. A.: Radium Therapy in Polypoid Ethmoiditis, Radiology **34**:449 (April) 1940.

151. Dalmark, G., and Goldin, J.: Shortwave Therapy of Chronic Sinusitis, Ugesk. f. læger **102**:303 (March 28) 1940.

152. Paterson, W. P. E.: The Treatment of Nasal Sinus Infection by Ultra-Shortwave Diathermy, Canad. M. A. J. **42**:454 (May) 1940.

represent the usual number of patients who get well spontaneously. However, if irrigations are fewer than otherwise, the treatment may be worth while. S. S.]

Chudoba [153] presents a highly technical explanation of the differences between long and short wave currents and elaborates on the details of the construction of the particular machine he employs. The one principle to be kept in mind with short wave therapy is that the stronger and deeper the action desired, the larger must be the electrodes and the greater the distance from the body. For instance, if he wants to cure ethmoiditis, he uses larger electrodes and goes farther away from the body, but he clamps the protective glass plates more firmly to the skin. When he treats empyema of the antrum, he also uses large electrodes, and in some cases he has used small electrodes in combination with these. The smaller ones are put near the diseased side if possible and the larger ones on the opposite side. The duration of the treatment is about thirty minutes, which he never oversteps. Treatments are given daily or every other day. The author used this treatment in 30 cases, 11 of which were cases of acute empyema of the antrum. After six to eight applications of the short wave therapy for twenty-eight to thirty minutes daily or every other day, absolute cure resulted in every case. In 3 cases of bilateral acute empyema of the antrum, after five to eight applications absolute cure resulted in eleven days. In every case an antral puncture was done before and after treatment, for diagnosis. Twice short wave therapy was used for chronic empyema of the antrum. In 1 case six daily applications, each for thirty minutes, were used without appreciable success. The patient reported abatement of his symptoms; nevertheless, a Caldwell-Luc operation was performed. In the second case eight applications of thirty minutes each were given every other day without success. In this case an operation was proposed, which the patient refused. Eight patients with ethmoiditis were treated; in 6 of these the involvement was unilateral and in 2 bilateral. In these cases tamponade with cocaine and epinephrine–treated gauze was added to the short wave therapy. In 7 cases a perfect cure resulted after one week. Three times the short wave therapy was given after an ethmoidectomy, with cure in two weeks, after seven applications. In 3 cases of acute rhinitis in which roentgen evidence of one-sided obliteration of the antrum was obtained by Fränkel's method, a cure resulted in eight to ten days. The author concludes that on the basis of his experience with this type of therapy it can be said that the effect is always favorable and that the treatment results in early abate-

153. Chudoba, V.: Shortwave Therapy of Inflammations of the Nasal Sinuses, Časop. lék. česk. **79**:220 (March 8) 1940.

ment of the subjective symptoms and leads to a more rapid cure of the conditions than does treatment by any other method.

Wahrer [154] has found short wave therapy of value in relieving pain and discomfort in acute and subacute involvement of the sinuses but cautions that it must be used in conjunction with the recognized measures for drainage and ventilation. He believes that it helps to shorten the course of sinal disease as well. He has not found it of much value in chronic involvement.

Scholtz [155] claims good results in acute involvement from steam baths (total), the steam being impregnated with the natural oils arising from fresh pine wood placed in the kettles. He also uses short wave therapy. In many cases of chronic involvement, he has been able to keep the patient comfortable with this therapy even when an operation was indicated, thus enabling the subject to carry on with his duties and avoid being incapacitated.

CLINICAL REPORTS

General Observations.—Wells [156] concludes from statistical studies that the average person averages two and one-half colds per year. He deduces certain fundamentals, which he states as follows:

(1) Whenever the nasal cavity is attacked by an acute inflammation there is a decided tendency for one or more of the paranasal sinuses to become involved. (2) The nasal sinuses are exceptionally prone to bacterial infection. [This is due to obstructions, impaired ciliary activity in the presence of colds and swelling of the ostiums S. S.] (3) There seldom occurs an isolated infection of one sinus alone: sinuses belonging to the anterior group are generally affected together; likewise the posterior group. (4) Sinus inflammation is essentially one of the mucous membrane—the bone, comparatively speaking, being rarely and slightly affected. (5) Pain is relatively rare in inflammation of the sinuses with the exception of the frontal, and when present is misleading as regards localization. (6) Profuse mucopurulent discharge accompanying a coryza is positive evidence of an acute sinusitis; and intermittent unilateral purulent discharge is characteristic of chronic sinus inflammation. (7) A considerable postnasal discharge or dropping is characteristic of the posterior sinus group. [Not always; it may be due to the antrum. S. S.] (8) The maintenance of free communication between the nose and paranasal sinuses is an essential requisite in the treatment of all sinus inflammation, acute and chronic. (9) Indications for treatment in acute sinus are threefold: the relief of pain, the combating of infection, and securing drainage and ventilation. (10) Indications for treatment of chronic sinus are the removal of products of inflammation, correction of local abnormalities which interfere with normal function, eradication of hopelessly diseased tissues and correlation of general conditions which act as predisposing causes.

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154. Wahrer, F. L.: Shortwave Therapy in Nasal Sinus Disease, Arch. Phys. Therapy **21**:410 (July) 1940.

155. Scholtz, H. G.: Was kann man mit konservativer Behandlung bei Nebenhöhleneiterung erreichen? Deutsche med. Wchnschr. **66**:852 (Aug. 2) 1940.

156. Wells, W. A.: The Fundamentals of Sinusology, M. Rec. **151**:159 (March 6) 1940.

Holding [157] challenges the defeatist attitude evidenced by some recent writings in the literature regarding the cause and treatment of colds and submits some common sense observations. Mainly the thesis dwells on the inciting factors of chilling of the body, the presence of a filtrable virus and improper living conditions and submits as curative mediums prolonged hot baths, a warm bed, rest, fluids, the early use of belladonna "internally in some form" and prolonged nasal douches with a hot saline solution. He expresses the belief that sleeping in cold drafty rooms is unhealthful, and he makes a plea for better ventilation, humidification and control of the temperature of the air of rooms in which one works and lives. On the whole there is much food for thought in this short paper, which cuts across the scientific point of view and comes down to the clinical experience of the ages, which in the main has been successful.

Bryant [158] presents a classification of types of sinusitis, with the usual findings in each class. After enumerating the factors which make up the protective mechanism of the nose, he points out that in chronic sinusitis there has been a weakening of one or more of the links in the chain due either to local or to constitutional causes, and that successful therapy depends on the proper recognition and correction of these factors. During a four year period not less than 400 patients with acute sinusitis were seen, all of whom recovered under simple palliative treatment except 8 with complications, 3 of whom died. In case 1 there was a fulminating infection of the frontal and ethmoid sinuses, with high temperature, local redness and edema, and cellulitis of the periorbita, following swimming. Incision and drainage were ineffective. The patient died twelve hours after admission to the hospital. Autopsy revealed acute cerebral edema, thrombosis of the jugular bulb, bilateral purulent sinusitis and edema of the scalp. In case 2 also the sinusitis followed swimming too soon after an acute head cold. Edema of the forehead and eyelids was present. There was a history of previous irrigations and treatment. Simple external incision and drainage were done. When the frontal sinus was opened, pus was found under pressure. There was osteomyelitis of the posterior wall, and the dura was covered with granulation tissue. There were also multiple brain abscesses. The patient died. Case 3 was that of a woman aged 40. Six years previously several of her teeth had been extracted, and she had had nasal symptoms ever since. During a severe cold she expectorated bloody sputum. Her left eye was blind; her nose was blocked by swelling of the turbinates and by pus. An external opening was

157. Holding, A. F.: The Common Cold and Common Sense, Laryngoscope **50**: 1012 (Oct.) 1940.

158. Bryant, F. L.: Paranasal Sinusitis: Factors in Management and Prognosis, Journal-Lancet **60**:273 (June) 1940.

made into the frontoethmoid region. Her condition grew progressively worse until she died. Autopsy showed multiple emboli and infarcts in the lung. Five other patients with a severe form of the disease recovered after conservative surgical treatment. In the histories of patients with chronic sinusitis Bryant found that weather conditions and allergy were prominent factors.

Teed [159] lists the physiologic factors involved in the protective mechanism of the nose as follows: the cilia lysozym, nasal respiration and endocrine influences. These are discussed and authorities cited. Additional biologic factors are allergy and the influence of bacteria. The latter factors are discussed and measures suggested to combat their untoward influences. The author has had good results with the pathogen-selective type of vaccine described by Solis-Cohen and Rubenstone. The organisms are taken from the nose or sinuses and grown in pure cultures. Intradermal tests are made with the pure cultures, and the vaccines made up of those to which the patient reacts. He believes the best results from roentgen therapy are obtained in cases of long standing in which the membranes are thickened. The last general factors in sinal disease which he cites are chilling, climate, diet and fatigue. He discusses these factors as possibly contributing to the incidence of sinal disease, through their influence on the functioning mucosa.

Capps's [160] article deals mainly with the tonsils. He cites Carl M. Anderson's series of 400 cases. However, in a group of 200 patients with sinusitis who did not have foci of infection other than those in the sinuses, only 3 had rheumatism and 1 bronchitis. Of another group of 200 who had foci of infection elsewhere, 26 per cent showed complications which may have been due to these infections elsewhere. He says the teeth, tonsils and prostate gland are more important as possible foci of infection than are the sinuses.

Blegvad [161] states that chronic sinusitis can be diagnosed and treated reliably only by a specialist. The general practitioner should consider the possibility of chronic sinusitis if the following conditions are present: (1) prolonged discharge of pus from the nose, particularly if it is unilateral; (2) offensive odor from the nose, especially a fetor, which now and then is noted by the patient himself, not by others; (3) symptoms from the rhinopharynx, particularly hypersecretion, which does not disappear in response to general treatment for pharyngitis; (4)

159. Teed, R. W.: Chronic Sinus Disease: Biologic Factors, J. Michigan M. Soc. **39**:772 (Oct.) 1940.

160. Capps, F. E. W.: The Faucial Tonsils and Accessory Sinuses of the Nose in Relation to Focal Sepsis, Post.-Grad. M. J. **16**:34 (Feb.) 1940.

161. Blegvad, N. R.: Inflammation in Accessory Nasal Sinuses, Ugesk. f. læger **102**:85 (Jan. 25) 1940.

frontal headache for which no other reason can be found (nasal stenosis, supraorbital neuralgia, infiltration around the cucullaris, refraction anomalies). He points out, however, that chronic sinusitis may develop without much secretion from the nose.

Headache.—Moore [162] believes that there is a misconception among lay persons that headaches mean sinal involvement, and that many people who have no sinal disease are taking nasal treatments for headache. He divides headaches of nasal origin into the following groups: (1) those due to obstruction (no infection); (2) those due to sinal infection; (3) those due to tumors; (4) those due to neuralgia. Group one includes headaches caused by the pressure of a markedly deviated septum and vacuum headache, which may come from the obstruction of sinuses other than the frontal. Headaches due to acute sinusitis are usually in close relationship to the affected sinus, are aggravated by stooping and are associated with a history of acute infection of the upper respiratory tract. Headaches in patients with chronic sinal disease are less frequent. The author describes the Sluder syndrome and nasociliary neuralgia. The cause of the latter may be anywhere along the course of the nerve. He quotes Nussun, that some patients with this condition have dryness and redness of the middle turbinate.

Alemán Caballero [163] presents a discussion of vacuum headache and the Sluder syndrome in connection with a case in point. Severe headaches and pains in the eye followed a fracture of the nose which resulted in a deviation of the septum. He claims that the trigger areas of the nasal mucosa were disturbed, causing the reflex vasomotor and sympathetic disturbances, and refers to Alden's plan of taking roentgenograms before and after treatment with epinephrine, showing the changes which shrinking of the mucosa produced.

Wilson [164] attempts to evaluate headache in relation to nasal disease but states that headache is not always a safe guide in diagnosis. He differentiates vacuum headache from that due to acute sinusitis by the fact that the former does not last as long and can be relieved completely with vasoconstrictors.

Gable [165] directs attention to pain and headache from chronic disease of the ethmoid sinus due to irritation of the branches of the sphenopalatine ganglion. Ocular complications of an inflammatory nature and

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162. Moore, P. M., Jr.: Headaches of Nasal Origin, M. Clin. North America **24**:311 (March) 1940.

163. Alemán Caballero, J.: Syndrome de Ewing-Sluder. Comentarios a unos casos clínicos, Semana méd. españ. **3**:974 (Aug. 3) 1940.

164. Wilson, T. G.: Headache of Nasal Origin, Irish J. M. Sc., January 1940. p. 38.

165. Gable, N. W.: Ethmoiditis, J. Florida M. A. **26**:498 (April) 1940.

some cases of bronchospasm are mentioned also. He notes the difficulty of making a diagnosis by transillumination or by roentgen examination. Frequent colds, with turgescence of the turbinate, are a common finding. He advises the Proetz displacement treatment, with the application of a 0.5 per cent solution of ephedrine and tampons impregnated with mild protein silver.

Jacques [166] cites a series of cases in which headache in the glabellar region, often with local tenderness, recurred daily morning and evening. Most of the patients were young neurotic females. There was usually a pale tumefaction of the middle turbinate and septum. Relief was obtained by cauterization. He believes the condition is due to chronic nonsuppurative involvement of the ethmoid sinuses, with responsive vasomotor reaction.

Love [167] describes a complex which occurs in a patient previously healthy. This consists of sudden attacks of vertigo, often coming on in the morning as soon as the patient gets out of bed. Rising suddenly or moving the head rapidly causes rotary vertigo, often with slight nausea and nystagmus. The symptoms are less marked later in the day. They are seldom found in the presence of an acute cold but may come on later. One often finds the postethmoid region to be tight. An attack can be elicited by giving a Proetz displacement treatment, but the attacks become milder with succeeding treatments and finally disappear. The author claims that the attack brought on by a Proetz treatment is diagnostic. He uses neo-synephrin hydrochloride for treatment every three to five days. He believes that the explanation of the complex is to be found in irritation of the vidian nerve, which may lie in the floor of the sphenoid sinus and which, by way of the great superficial petrosal nerve and the geniculate ganglion, may connect with the vestibular ganglion. He found records of 52 cases in the literature from 1929 to 1935 and describes 2 cases which he observed.

Maxillary Sinus.—Brunner and Wall [168] report a case in which fatal hemorrhage followed antral puncture. A man aged 54 had soreness over the right side of the nose for three weeks and severe pains on the right side of the face and forehead. There were weakness and general muscular pain and later puffiness of the right eye and diplopia. Examination revealed paresis of the external oculomotor nerve on the right,

166. Jacques, P.: Syndrome névralgiforme de la glabelle, Ann. d'oto-laryng., September-October 1939, p. 797.

167. Love, T. S.: Nasal Vertigo—A Clinical Entity Often Overlooked, Dallas M. J. **26**:4 (Jan.) 1940.

168. Brunner, H., and Wall, J. W.: Carcinomatosis of the Nasal Mucous Membrane (Fatal Hemorrhage After Puncture of the Maxillary Sinus), Ann. Otol., Rhin. & Laryng. **49**:688 (Sept.) 1940.

slight exophthalmos, sensitivity of the conjunctiva and clouding of the antrum and frontal sinus. Roentgen examination confirmed these observations. The pupils were normal. Puncture of the right antrum through the inferior meatus yielded a small amount of pus and was followed by much bleeding. At the time of puncture there was an impression as of a soft tumor-like mass in the antrum. There were later evidences of loss of blood, presumably internal, as there was not much blood coming from the nose. Despite repeated transfusions and supportive measures, the patient became progressively weaker and died twenty-four hours after the puncture. Autopsy revealed a large carcinoma of the stomach and general carcinomatosis of the dura. There was no gross tumor in the sinus. The authors believe that the gastric hemorrhage was induced by reflex irritation of the vagus nerve from injury to branches of the trigeminus nerve in the antrum.

Berke [169] reports several cases which show that maxillary sinusitis of which the patient is unaware may cause pain in the teeth, often leading to unnecessary work on these structures without relief. The condition is characterized by sensitiveness of the upper posterior teeth to percussion, pain of varying intensity, generally confined to the affected maxilla, and rarely swelling of the cheek. Appropriate roentgen studies and investigation of the sinus should insure the correct diagnosis, and relief is readily obtained by attention to the sinus. In 1 case pus from an infected sinus had burrowed through the thin outer wall of the antrum and become localized in the mucobuccal fold of the first molar area. In 2 other cases the infection caused swelling of the cheek.

Haugh [170] states that the implantation of teeth in the paranasal cavities is relatively rare and that the detection of the structures is not often an easy matter. This condition was diagnosed in patients with entirely different diseases, as illustrated by the following case. A 12 year old girl came for observation, complaining of frequent sore throat and occasional aching in the joints. At 4 years of age she had a few contagious diseases and was operated on for otitis media. Her nose was discharging, and she had frequent colds and headaches. Examination revealed nothing of diagnostic significance except poor development, chronic tonsillitis and a sticky milky excretion from the middle meatus of the nose. Roentgenograms showed density of each maxillary cavity and the shadow of a tooth (one on each side), the crown of which was in the alveolar process. On tapping of the maxillary cavities, foul pus

169. Berke, J. D.: Maxillary Sinusitis and Obscured Partial Impaction as Causes of Dental Pain of Undetermined Origin, J. Am. Dent. A. **27**:57 (Jan.) 1940.

170. Haugh, C.: Case of Tooth Grown into Antrum of Highmore, Gyógyászat **80**:33 (Aug.) 1940.

was obtained. A Caldwell-Luc operation on the left side revealed the usual pathologic changes of the mucosa of the cavity and a very thin bony wall separating the cavity in the sagittal plane. A healthy canine tooth was taken out (without any chance being given to manage the case by routine orthodontic means). The author states that the tooth removed was in the normal position but that there was some disproportion of space between the neighboring teeth and that he did not expect the tooth to descend into its proper place. He explains further that the purulent process of the maxillary cavity, not the tooth in question, was the indication for the operation.

Dental Infection.—Ottoni de Rezende [171] discusses in a general way various theories of the origin of dental cysts, quoting many authorities, and the relationship of dental infection to infection of the antrum. He points out that a negative result from irrigation does not rule out sinal disease. In many cases in which disease is latent, a sinus acts as a focus of infection, due to extension of disease of the bone from the paradentium, which can be disclosed only by careful roentgen studies and cooperation with an oral surgeon.

The case reported by Sato [172] was that of a woman of 56 with delayed healing of a fistula after extraction of the upper second molar tooth on the left side. A sound could be passed through the fistula into the antrum. Roentgenograms showed cloudiness of the antrum. Irrigation yielded foul brownish secretion. Cure followed repeated irrigations.

Frontal Sinus.—Routine examination by Eldridge and Holm [173] of 200 successive female patients admitted to St. Elizabeth's Hospital, Washington, D. C., revealed roentgen evidence of hyperostosis of the frontal bone in 25 per cent. However, the characteristic "triad of Morel" was not so frequent. Obesity was present in only 8 cases, visual defects in none and headache in only 3, and there were none of the other characteristic neurologic disorders usually associated with this syndrome. The authors express the belief, therefore, that the fact that the osseous changes were twenty times as common in patients with mental disease as in the general population constituted a significant though at present unaccountable finding, but the absence generally of associated symptoms shows that this finding alone is not sufficient for a diagnosis of the Morel-Morgagni disease.

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171. Ottoni de Rezende, M.: Relacoes patologicas entre os dentes y os antros maxillares, Rev. brasil. de oto-rino-laring. **7**:463 (Nov.-Dec.) 1939.

172. Sato, K.: Ueber einen Fall von Kieferhöhlenempyem das als rein odontogen anzusehen ist, Oto-rhino-laryng. **13**:24 (May) 1940.

173. Eldridge, W. W., and Holm, G. A.: The Incidence of Hyperostosis Frontalis Interna in Female Patients Admitted to a Mental Hospital, Am. J. Roentgenol. **43**:356 (March) 1940.

Hemphill and Stengel [174] present a brief review of the history and findings in 3 cases of Morgagni's syndrome. Case 1 was that of a woman of 31 with rapidly developing dementia, symptoms of pyramidal disorder and ataxia and hypersensitivity of the whole body, especially of the lower extremities. Roentgen examination of the skull showed great thickening throughout, especially marked in the frontal and parietal regions. Case 2 was that of a woman aged 65, who had experienced psychotic changes for twenty years but had had melancholia for only six months. There were organic changes in the nervous system, symptoms of meningeal irritation and loss of the reflexes in the lower limbs. During the previous six months she had had severe headaches. Roentgenograms showed marked hyperostosis frontalis interna. Case 3 was that of a woman aged 32, with paranoid symptoms, virilism and menstrual disturbances. The tendon reflexes were exaggerated and the blood count slightly raised. Roentgenograms showed hyperostosis similar to that of the patient in case 2 (her mother). Autopsy in case 2 revealed diffuse hyperostosis of the cranial vault, especially in the frontal and parietal regions, with nodular frontal endostoses. The bone was eburnated, the diploe reduced and the dura thickened. There were flattening and atrophy of the frontal lobes, hypertrophy of the adrenals and degenerative changes in the brain. The anterior lobe of the pituitary gland showed a large number of acidophilic cells, the arrangement of which was suggestive of adenoma. There was marked proliferation of the connective tissue. The parathyroid glands showed pathologic changes; three of the four were very small; there was an increase of oxyphilic cells, and one gland consisted entirely of these cells. There was some atrophy of the thyroid gland. Hemphill and Stengel suggest that the variety of forms of mental disorder seen in these cases is "due to interaction between the mental constitutions of the individuals and the disorder of the brain and endocrine glands."

The case reported by Roger, Schachter and Boudouresques [175] was that of a woman of 58 with generalized obesity, slight hairy virilism and marked melancholia with suicidal tendency. Roentgenograms showed typical thickening of the posterior wall of the frontal bone. The authors present a table of symptoms for comparison with the syndrome of acromegaly, Cushing's syndrome and the syndrome of Morgagni, all of which have points in common. They believe the condition is due to hyperfunction of the anterior lobe of the pituitary gland and cite Martimor's experi-

174. Hemphill, R. E., and Stengel, E.: Morgagni's Syndrome: A Clinical and Pathological Study, J. Ment. Sc. **86**:341 (May) 1940.

175. Roger, H.; Schachter, M., and Boudouresques, J.: Un nouveau cas de syndrome de Morgagni chez une mélancolique, Gaz. d. hôp. **113**:133 (Feb. 21) 1940.

mental work on rats, in which injections of extracts of the anterior lobe of the pituitary gland produced changes of the cranial bones similar to those observed in osteitis frontalis interna.

Belloni [176] reports a case of hyperostosis frontalis interna which was unusual because of the occurrence of attacks typical of Ménière's disease, vertigo, vomiting and marked nystagmus, with diminution of hearing and impairment of vestibular responses in the intervals. The patient was a woman of 61 whose roentgenograms showed, in addition to the hyperostosis, a rather large pituitary gland and marked impressions of the venous channels but no other evidence of increased intracranial pressure. The apex of the petrous bone and the internal auditory meatuses were normal. Belloni accounts for the attacks on the basis of vasomotor crises due to endocrine disturbances, since the symptoms came on after the menopause, which preceded by several years the diagnosis of hyperostosis frontalis interna.

The case reported by Calabrese [177] was that of a man of 33 with a pituitary syndrome of polyuria, adiposis and loss of sexual power, who showed typical thickening of the inner table of the frontal bone; the sella turcica was of normal size, but the clinoid processes were hypertrophied, and there was ossification of the interclinoid ligaments.

Richter [178] cites 9 cases and presents roentgenograms. After discussing the literature and cases which he has encountered, he concludes that the chief factor in the osseous hyperplasia is a hormonal disturbance arising chiefly from the pituitary gland, since the condition is most often observed in women at the climacteric and in men who are advanced in years. As to why the internal layer is affected rather than the external, he offers no hypothesis. He emphasizes the importance of roentgen studies in explaining many mental disturbances and the other symptoms characteristic of fully developed hyperostosis of the inner table of the frontal bone.

Botelho [179] presents a case of hyperostosis frontalis interna which he believes is the first of its kind reported from Brazil. He mentions slight acidosis and hypercalcemia as characteristic. The syndrome consists of neuropsychosis with obesity, vertigo and epileptiform attacks, often with transient hemiparesis and a tendency to depression. In the case reported

176. Belloni, G. B.: A proposito dei disturbi cocleo-vestibalari nella iperostosi frontale interna (endocraniosi di Morgagni), Riv. di clin. med. (supp. Margin. otolaryng.) **40**:68, 1940.

177. Calabrese, G.: Un nuovo caso di endocraniosi iperostosica tipo Pende, Riforma med. **56**:675 (May 25) 1940.

178. Richter, L.: Zur Hyperostose des Stirnbeins, Röntgenpraxis **11**:651 (Dec.) 1939.

179. Botelho, T.: Hiperostose frontal interna, Rev. med.-cir. do Brasil **48**:1 (Jan.) 1940.

all the symptoms were present, but the calcemia was only slight. The rare features of the case were (1) the occurrence of the condition in a male and (2) the involvement of the occipital as well as the frontal bone. The patient refused an operation for removal of the parathyroid gland unless a cure could be guaranteed. However, the author believes sincerely that the operation may not have been successful.

Prieto and Llamosas [180] report a case in which late tertiary manifestations of hereditary syphilis occurred in a woman of 25. When she appeared in the clinic, she revealed a healed deforming lesion of the lower part of the nose, a large ulcerated area over the left frontal region, with dead bone exposed, a similar smaller lesion of the right frontal area and pulmonary lesions, such as consolidation, cavities and bronchiectasis. A large sequestrum was removed from the left frontal region, exposing the sinus, but despite active antisyphilitic treatment the woman died. Autopsy confirmed the serologic diagnosis. Photographs show unusually extensive lesions.

Frontoethmoid, Ethmoid and Sphenoid Region.—McCollough [181] presents a résumé of 42 cases of acute fulminating frontoethmoiditis encountered at the Eye and Ear Hospital, Pittsburgh, in a period of ten years. He defines the disease as "that type of acute suppurative infection in the ethmoidal and frontal sinuses which may, at any time within a few hours to a few days, produce definite clinical evidence that the disease has spread to some surrounding structure." The cases are divided into five groups according to the type or extent of the involvement of adjacent structures. It is interesting to note that in almost half the cases the offending organism was Staphylococcus aureus. Preoperative complications observed were meningitis (2 cases), orbital cellulitis (6 cases), extradural abscess (3 cases) and osteomyelitis (3 cases). Twenty-seven of the entire group of patients recovered with palliative treatment, such as drainage, transfusion and chemotherapy; the remainder (45 per cent) required surgical intervention. The Lynch operation was done in 25 cases. The author believes that since the periosteum and bone of the anterior wall are not disturbed there is little danger of inciting osteomyelitis. He emphasizes the importance of being able to evaluate symptoms of spreading infection, so that appropriate treatment may be instituted in time.

In distinguishing between the usual exudative type of infection of the ear and the sinuses and the necrosing type, Richter [182] states the belief that the latter is due to a low state of resistance in the patient

180. Prieto, R., and Llamosas, N.: Gomas lueticos mutilantes naso-fronto-ciliar, An. oftal. y oto-rino-laring. d. Paraguay **1**:79 (Dec.) 1939.

181. McCollough, T. B.: Acute Fulminating Fronto-Ethmoiditis, Pennsylvania M. J. **43**:821 (March) 1940.

182. Richter, H.: Ueber nekrotisierende Eiterungen des Mittelohres und der Nasennebenhöhlen, Hals-, Nasen- u. Ohrenarzt (pt. 1) **31**:21, 1940.

rather than to any specific virulence of the invader. He cites 5 cases in which the ear spaces were involved and 2 in which the sinuses were involved. In all the infection was due to scarlet fever. He deduces that since all the symptoms were ameliorated after adequate local and general treatment his theory must be right. In the 2 cases of sinal infection cited, one that of a boy of 12 and the other that of a young man of 19, the symptoms came on during an attack of scarlet fever, and a subperiosteal abscess of the frontal bone was found, but no fistula; hence, the infection must have been transmitted via the perforating vessels. In both cases the frontal and ethmoid mucosa was necrotic and sequestrums were present in the ethmoid sinus. Both patients recovered despite severe constitutional symptoms. In the second case diphtheria organisms also were found but evidently did not contribute to the necrosing process. Specific serum was given.

Carruthers [183] considers ethmoiditis of two types, suppurative and polypoid. The former ordinarily is associated with infection of the antrums, and when adequate surgical treatment is given, usually is cured. The polypoid type frequently tends to recur despite adequate surgical treatment. He also finds that the suppurative type often is associated with bronchitis, whereas the polypoid form is connected with asthma or other allergic disorders. Forty per cent of his patients with polypoid ethmoiditis proved to be allergic. Positive skin tests were observed in 35 per cent of the group. On the other hand, of a series of 16 patients with hay fever and asthma, only 12 per cent had polyps. In this group positive skin tests were observed in 66 per cent. He says: "Does allergic disease or an allergic potentiality cause or predispose to polypi, or do polypi or some factors in their etiology or metabolism or in the site they commonly occupy tend to excite allergic reactions in susceptible individuals?" He inclines to the latter explanation. He also finds that endocrine factors have a part. Histologic studies of specimens removed showed in 26 cases distention of the glands, with accumulation of large numbers of leukocytes around the acini and ducts and beneath the surface epithelium. Most of these cells were of the lymphocytic and plasma type. Eosinophils were less numerous than is generally believed. The author suggests the possibility that a virus was the inciting factor in many of the cases. He claims that a virus has been proved to be the cause of certain myxomatous and fibrous tumors.

Kepes [184] claims that many intracranial infections have their origin in the sphenoid sinus and are not diagnosed until autopsy. Of all the sinuses, the sphenoid offers the best route to the cranial cavity and the one

183. Carruthers, D. G.: The Investigation and Diagnosis of Ethmoiditis, M. J. Australia **2**:147 (Aug. 17) 1940.

184. Kepes, P.: Latent Suppurations of the Sphenoid Sinus with Intracranial Complications, Pract. oto-rhino-laryng. **3**:65, 1940.

which often presents the fewest clinical symptoms. He divides the cases into three groups: (1) those in which the symptoms are the least and the fewest and in which for that reason usually no medical treatment is given; (2) those in which symptoms are present but are not characteristic of infection of the sphenoid sinus, and (3) those in which the symptoms of involvement of the sphenoid sinus are masked or overshadowed by symptoms of intracranial disease and in which the sphenoid complex is thus pushed into the background so as often to be overlooked. In the cases of group 1 often there is a latent chronic infection which has not been diagnosed but which becomes the seat of an acute exacerbation, with rapid extension to the cranial cavity, the acute process being of such duration as to escape detection because of the overwhelming intracranial complication. Conditions of this type have often been seen at autopsy. In the cases of the second group the sphenoid symptoms are definite and can be evaluated provided the surgeon takes the trouble to investigate thoroughly. The cases of the third group are those in which there is a concurrent otitic infection with intracranial symptoms, the usual inference being that the aural infection is the cause, whereas in many cases the spread has been from the sinus and not from the ear. A case in point is cited. A woman aged 30 was operated on for appendicitis; headache and fever developed postoperatively. The headache was diffuse. A few days later there were violent pains in the right ear. An aural examination gave negative results. The pains increased, and a few days later the drum membrane was inflamed. The manifestations were similar to those of myringitis bullosa. Paracentesis yielded a serous discharge. On the same evening chills and symptoms of meningitis were present. The spinal fluid was opaque. The mastoid process was opened, but nothing abnormal was seen. On the next day the patient died. Autopsy revealed pus in the arachnoid spaces at base of the brain, especially about the gasserian ganglion. The mucosa of the sphenoid sinus was swollen and covered with pus. There was a defect with a ragged border on the lateral wall. The author points out that the warning symptom of pain emanating from the gasserian ganglion was not properly evaluated and that the fact that the pains were referable to the ear was misleading, as these were really due to irritation of the vidian nerve and the sphenopalatine ganglion. He seeks to impress on one the fact that often a correct diagnosis is not made because the physician is more otologically minded than sinus minded when there are symptoms of intracranial involvement with aural symptoms.

Pond [185] reports a case of Paget's disease, pituitary tumor and empyema of the sphenoid sinus. A man aged 62 had severe headache,

185. Pond, C. W.: Paget's Disease, Pituitary Tumor and Abscess of the Sphenoid Sinus: Report of a Case, Ann. Otol., Rhin. & Laryng. **49**:500 (June) 1940.

deep dull boring pain behind the eyes, nasal obstruction, postnasal discharge and deviation of the septum. Roentgen examination showed blurring of the ethmoid and sphenoid sinuses. The vision was normal, and an operation on the septum was advised but refused. Fourteen months later the patient was seen again, and his condition was finally diagnosed as Paget's disease. Bony changes were seen in the skull. The sella turcica was eroded and the clinoid processes were gone. The posterior wall of the sphenoid sinus was not seen. The patient died of meningitis. Autopsy showed pus at the base of the brain; the pituitary gland was lying in a necrotic mass composed of the sella turcica, the clinoid processes and the roof of the sphenoid sinus; there was pus in the ethmoid sinuses and the antrums. The pituitary gland was about 35 mm. in diameter. The bones of the skull showed characteristic fibrous tra[illegible]e.

Monteiro [186] reports a case of cholesteatoma involving several sinuses in a man of 34. The symptoms were nasal discharge and a fistula in the gingival recess, communicating with the antrum. Previously the patient had been operated on twice. The nasal fossa was filled with a yellow-white mass, which had a foul odor. There were granulations, which bled easily. Roentgen examination revealed erosion of the external and internal walls of the antrum. A Caldwell-Luc operation revealed a cholesteatoma mass filling the maxillary, ethmoid and sphenoid sinuses. The nasal floor was eroded as well. The frontal bone was uninvolved. The cholesteatoma was removed and the resultant large cavity was packed and was irrigated daily with hydrogen peroxide. After three months a plastic operation was done, to close the fistula into the mouth. The author cites the opinions of several authors as to the cause of the condition. He believes that cholesteatoma is due to metaplasia of the normal epithelium into pavement epidermis by ingrowth from without, pressure of retained secretions and inflammatory reaction.

Childrey [187] reports several cases of Paget's disease. A man aged 24 had poor vision of the left eye. His face was asymmetric, with bulging of the forehead, and he was myopic. With glasses his vision was 70 per cent in the left eye. The fundi were normal. The left eye was 12 mm. above the plane of the right. Roentgenograms revealed marked hyperostosis and sclerosis of the bones of the anterior and middle fossae, with great thickening of the bones of the supraorbital region. There were involvement of the horizontal frontal plate and increased density of the ethmoid labyrinth; the sphenoid was practically

186. Monteiro, A.: Colesteatoma do seio maxilar do esfenoide y das celulas etmoidas, Acta med., Rio de Janeiro **5**:13 (Jan.) 1940.

187. Childrey, J. H.: Paget's Disease of the Bones of the Skull with Obliteration of the Sinuses, Arch. Otolaryng. **31**:333 (Feb.) 1940.

solid bone with no evidence of a sinus. Both optic foramens were very small. Involvement of the bones was more advanced on the left side than on the right. Apparently there was no left frontal sinus, and there was little, if any, air space in the ethmoid labyrinth. No other parts of the skeleton were involved. There were both osteoporosis and osteosclerosis. There was a possibility of trauma at birth, through forceps delivery. A woman aged 45 had reduced vision, that of the left eye being limited to ability to see fingers at 2 feet (61 cm.); that of the right eye was reduced to 30 per cent. There was bilateral posterior staphyloma and changes in the maculas. The visual fields were contracted, and there was central blurring of the left eye. Roentgen examination of the skull revealed characteristic thickening, mostly in the parietal areas. A woman aged 59, a short squat person, whose back was bent and whose shoulders sagged, had failing vision in both eyes. Her head and the upper part of her face were large. Vision in the right eye was 5 per cent and that in the left 10 per cent. There were opacities in the lenses, temporal pallor of the disks, generative macular changes and evidence of hemorrhage. There was bilateral contraction of the visual fields. Roentgenograms showed thickening and a general wooly appearance of all bones except the mandible. The sinuses were obliterated, as were the mastoid cells. The upper cervical vertebrae were involved. There were deposits of dense sclerotic bone. Roentgen examination of the spine, pelvis and most of the long bones showed advanced Paget's disease.

Goodyear [188] reports a rare case of tularemia. A woman aged 27 possibly had acute involvement of the sphenoid sinus eight days before examination. She had severe frontal and occipital headache on the right side, slight vertigo and chills. Her temperature was 104 F. There were postauricular adenitis and conjunctivitis on the right side. The right side of her nose was swollen and red. Sulfanilamide was given, but the pain continued. She was seen by the author on the eighth day. The right eye was red and swollen and the eyelids thick, with yellow spots on the red conjunctivas. The glands behind the right ear were swollen. The nasal mucosa was pale. Roentgen and other examination revealed no evidence of sinal disease. There was pain in the right eye, in the temporal region and in the shoulder. The patient lived on a farm. Eleven days previously she had dressed wild rabbits, but she stated that she wore rubber gloves. The symptoms appeared three days later. On the eighth day an agglutination test was positive. The diagnosis was oculoglandular tularemia. Serum was given. Forty-eight hours later the temperature receded, and

188. Goodyear, H. M.: Tularemia: Assimilating Symptoms of an Acute Ethmoid, Sphenoid Infection, J. Med. **21**:81 (April) 1940.

recovery ensued. The author directs attention to the absence of sinal findings despite typical headache and the characteristic yellow spots on the red conjunctivas, early signs of tularemia.

Fractures and Foreign Bodies.—Dechaume [189] directs attention to the fact that in some cases a fracture of the upper jaw frequently escapes detection in roentgenograms because the fracture is incomplete or the displacement is insufficient to cause deformity. Yet these cases may be complicated by symptoms referable to the maxillary sinus, developing at a later date. When such a fracture penetrates the mucosa of the sinus, one may see emphysema of the tissues of the cheek, hemorrhage into the sinus, subsequent suppuration and occasionally anesthesia due to pressure on the infraorbital nerve. He cites 2 cases in point, in which it was necessary to open the sinus for drainage. He points out that cloudiness of the antrum shown by roentgenograms is suggestive of fracture, especially if there is no history of a preceding infection. In one of his cases a second roentgenogram showed the fracture line, which in the beginning was obscured by edema.

Gottesberge [190] reports an interesting case. A few minutes after diving from a 15 foot (450 cm.) board the patient noticed a swelling of both eyelids on one side, which was not especially painful. Examination revealed emphysema of the soft tissues, marked exophthalmos and impaired mobility. The author claims that the symptoms were caused by fracture of the lamina papyracea. It has been proved experimentally that this may result from trauma to the skull from a blunt object, especially in the supraorbital or the occipital region. Experiments have shown that a pressure of 10 mm. of mercury is all that is necessary for the production of orbital emphysema and 50 mm. for orbitopalpebral emphysema. He cites a similar case reported by Steurer.

Hargett [191] records the spontaneous expulsion of a piece of gauze from the antrum by several irrigations through the inferior meatus. The patient had a molar tooth extracted, and evidently the packing placed in the alveolar socket was pushed into the antrum.

The case reported by Golden [192] was that of a boy of 13 who four days previously was accidentally shot in the left eye with a single buckshot from an air rifle at ten feet (300 cm.). There was a punctate

189. Dechaume, M.: A propos des fractures incomplètes du maxillaire supérieur et de leurs complications sinusiennes, Presse méd., **48**:627 (July 31-Aug. 3) 1940.

190. zum Gottesberge, A. M.: Orbitales Emphysem durch Fraktur des Siebbeins nach Kopfsprung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **147**:66, 1940.

191. Hargett, E. R.: Expulsion of Foreign Body from the Antrum with the Aid of Irrigation, Arch. Otolaryng. **31**:332 (Feb.) 1940.

192. Golden, M. G.: Air Rifle Buckshot in Left Posterior Ethmoids: Operative Removal, Ann. Otol., Rhin. & Laryng. **49**:1032 (Dec.) 1940.

wound of the sclera at the level of the canthus, midway between the inner canthus and the medial margin of the iris. Roentgenograms showed a foreign body in the region of the left orbit, localized in the left posterior ethmoid cells. With the patient under anesthesia induced with ether and with a posterior plug in place the middle turbinate was infracted. The ethmoid cells were entered intranasally. The bullet was found and extracted. There were no bad after-effects.

Miscellaneous.—Harris [193] describes the usual symptoms of allergy of the nose, with the observations hitherto often mentioned in the literature. Since infection may develop in association with the allergy, it is important not to miss the diagnosis and to avoid both unnecessary surgical intervention and subsequent infection, which only complicates the picture. Since the treatment of the condition consists of elimination, substitution or immunization, he presents a list of instructions for the patient, which should assist the physician in curing the condition: 1. Avoid all dust, which means the removal of woolen rugs, draperies and articles which may harbor dust. 2. Avoid contact with feathered or furred animals. 3. Avoid blooming plants, flowers, weeds, etc. 4. Avoid cosmetics containing orris. 5. Avoid insect powders and sprays. 6. Use kapok pillows and mattress. 7. Avoid cooking odors, paints, garage odors, etc. 8. Avoid drafts. 9. Take only tepid baths; no swimming. 10. Adhere to the prescribed diet. 11. Do not use nose drops other than as prescribed. 12. Avoid overexertion and fatigue.

Through observation of professional swimmers and through experiments on animals Mezz [194] found that despite holding of the breath water enters the nasal chambers and that this factor, not the chilling of the body, is responsible for changes observed in the nasal mucosa. He explains the entrance of water as due to insufficient closure of the nostrils and inadequate air pressure despite the taking of a deep breath prior to diving. It seems that the muscular exertion of diving is responsible for the expulsion of most of the inspired air even though the diver thinks he is holding his breath. The author presents a protective nose clip which has proved effective in preventing the entrance of water into the nose.

Crosson, Jones and Sayers [195] report on a device for the relief of "ear and sinus block" in men who work under compressed air. As is

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193. Harris, M. C.: Allergic Sinusitis, New York State J. Med. **40**:1020 (July 1) 1940.

194. Mezz, D.: Otorhinologic Sequelae of Swimming: Analysis of Present Concepts; a New Method of Prevention, Laryngoscope **50**:479 (May) 1940.

195. Crosson, J. W.; Jones, R. R., and Sayers, R. R.: Helium-Oxygen Mixtures for Alleviation of Tubal and Sinus Block in Compressed Air Workers, Pub. Health Rep. **55**:1487 (Aug. 16) 1940.

true of the tympanic cavity, the sinuses may be closed off by swelling of the mucosa around the ostiums, which causes severe pains in the face and head and leads to prolonged congestion and possible infection. For the relief of this condition the authors devised an apparatus for the worker, which he uses while in the man lock.

It consists of a small cylinder of 80 per cent helium and 20 per cent oxygen connected through a small reducing valve to a "lung" or breathing bag adapted from a 2-hour self-contained oxygen breathing apparatus frequently used in mine rescue work. The breathing bag is equipped with an admission valve which is automatically tripped when the bag collapses. Attached to the bag is a rubber tube on the end of which is a detachable face mask.

The theory is that since the helium replaces the nitrogen in the inspired air, by virtue of the fact that helium is more than twice as diffusible as nitrogen, the former will more quickly bring about a restoration of normal equilibrium between the enclosed cavities and the outside air than is otherwise possible and thus will prevent the unpleasant sequelae frequently encountered.

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Abstracts from Current Literature

Ear

ROENTGEN SIGNS OF MASTOIDITIS AND ITS COMPLICATIONS. V. C. JOHNSON, J. A. M. A. **115**:510 (Aug. 17) 1940.

Johnson discusses in some detail the varied roentgenologic signs of mastoiditis. He points out that for an accurate and thorough roentgen examination of the mastoid bone it is important to employ a careful technic not only for the mastoid process but also for the squamous and petrous portions of the temporal bone. Multiple exposures, each designed to portray some particular portion of the temporal bone, are essential. While a single exposure may outline just the pathologic process one wishes to see, no one can foretell what exposure will give that information.

In evaluating the roentgenograms representing detailed osseous anatomy, one must bear in mind that variations from the normal appearance, either unilateral or bilateral, may result from vagaries of development or from acquired disease. Exact anatomic details should be described to the otologist by the examiner and questions of cellularity, with the likelihood of deviations from the rule of bilateral symmetry, should be explained, thereby obviating the probability of a false interpretation. Precise reading and evaluation of roentgenograms of an acellular mastoid in relation to the opposite mastoid, when the latter is normally pneumatized, are important, as the roentgenologist can only report the extent of the process and gage the relative density of the bone in the diseased area.

Johnson comments on the roentgen findings in suppuration of varying degrees and duration, on the position of important blood vessels, such as the lateral sinus and the emissary veins, on cholesteatoma and the nature thereof, and a petrositis and the attendant extension of cellularity. Concerning the last he says that cellularity of the petrous bone is proportionate to the cellularity of the mastoid process. He comments also on osteomyelitis, brain abscess and tumors.

In conclusion he emphasizes once more the importance of a precise and careful technic of roentgen examination and of sound judgment in the evaluation of roentgen findings.

GORDON, Philadelphia.

OTONEUROLOGIC DIAGNOSIS OF CEREBRAL TUMORS. W. SALEM, Rev. argent. de oto-rino-laring. **9**:53 (March-April) 1940.

Salem, on a basis of 30 cases in which the presence of cerebral tumor was proved, either by autopsy or by operation, discusses the importance of the vestibular test in localizing intracranial tumors. After giving a brief anatomic résumé of the vestibular and cochlear pathways, from the internal ear to the central nervous system, he outlines the method of examination by vestibular and hearing tests and stresses the significance of the findings from these tests.

As a result of his study he concludes: (1) that the labyrinth is of great value in localizing intracranial lesions but of less value in cases in which there is a marked increase of intracranial pressure; (2) that an isolated cerebellar tumor as a rule cannot produce spontaneous nystagmus; (3) that caloric hyperexcitability, with large and irregular nystagmus, has no value in localizing a lesion in the posterior fossa; (4) that progressive clinical signs of intracranial tumor, without involvement of the cochlear-vestibular system, can generally be considered to indicate that the lesion is outside of the posterior fossa, and (5) that postcaloric signs other than nystagmus are of great diagnostic importance.

PERSKY, Philadelphia.

SURGICAL INDICATIONS IN CHRONIC MASTOIDITIS. ATILIO VIALE DEL CARRIL, Rev. argent. de oto-rino-laring. **9**:69 (March-April) 1940.

Del Carril discusses the relative merits and limitations of treatments for chronic suppuration of the mastoid. He believes that conservative treatment should be employed when the process has not produced too extensive a lesion of the bone. Medical (or nonsurgical) treatment is of the first order, but if after a sufficient therapeutic test a cure is not produced, surgical measures are indicated.

He discusses the various surgical measures, discrediting minor surgical procedures, such as ossiculectomy, and even the modified radical operation. He compares the relative merits of the endaural and the retroauricular approach in doing the complete radical mastoidectomy, which is his method of choice. He definitely favors the latter approach.

His indications for a complete radical mastoidectomy are: (1) failure of intensive medical treatment; (2) cholesteatoma; (3) involvement of the labyrinth, and (4) premonitory signs of a cerebral or cerebellar involvement (such as temporal or occipital headache, stupor or bradycardia).

He concludes by stating that he does not believe that the endaural route gives sufficient exposure and that, in consequence, he favors the retroauricular approach.

PERSKY, Philadelphia.

EXCESSIVE NOISE AND OTALGIA. SEIICHI KAWATA, Oto-rhino-laryng. **13**:525 (Aug.) 1940.

Kawata reports 4 cases of otalgia observed among the workers in airplane plants.

CASE 1.—A man aged 25 years complained of bilateral otalgia, slight impairment of hearing and tinnitus of three months' duration. He spent three hours daily testing the performance of airplane engines and the remainder of his working hours in close proximity to the noise. At first he noticed tinnitus, and three months later otalgia developed, which was felt as though an external pressure were being applied on the tympanic membranes. Otalgia was continuous, being slight in the morning but progressively worse toward the afternoon and evening. His hearing became gradually impaired. Audiometric examination disclosed a loss of 20 decibels up to 512 cycles in both ears.

CASE 2.—A boy aged 17 years complained of bilateral otalgia, which began seven months after he started work in the test room. Tinnitus was noticed about an hour after each day's labor. An audiometric test showed a loss of 20 to 40 decibels, most marked between 512 and 1,024 cycles.

CASE 3.—A boy aged 19 years complained of severe otalgia after each day's work. This symptom began after he had worked eleven months in the test room. The pain was sharp and shooting in character, lasting thirty minutes, and was worse on the left side. The right eustachian tube was normal, but the left was obstructed. The hearing was normal except that there was a drop on the left.

CASE 4.—A boy aged 17 years complained of bilateral otalgia at the end of each day's work after five months' service. There was no tinnitus. Abnormal conditions were not noted in the ear, nose or throat except a slight congestion over the handle of the malleus on the right. An audiometric test showed a slight loss of hearing in 512 and 4,096 cycles.

All the patients but the last were treated with subcutaneous injections of vitamin B_1, and the results were most satisfactory.

Kawata states that there are dual sources of noise in the airplane—one is from the engine and the other from the propeller. The engines rotate at the rate of several thousand times per minute and the propellers at 2,000 per second, but the combined effect of the two different types of noise is most complicated. The relation of otalgia of similar character due to exposure to similar types of noise in these 4 cases is most interesting. The first 2 patients suffered from

continuous pain, though the time of onset of pain appears to have no relation to the length of the exposure. The impairment of hearing was generally slight, though the condition of the patient in the first case is suggestive of occupational deafness.

HARA, Los Angeles.

PNEUMOCOCCIC TYPES OF MIDDLE EAR SUPPURATION. B. R. HANSEN, Acta otolaryng. **28**:340, 1940.

Research on the bacteriology of acute suppurations of the middle ear is presented, with special reference to two points: (1) the introduction of chemotherapeutic agents in recent years and (2) the enlargement of serologic bacteriology, with special reference to pneumococci of various types and the possibility, because of typing, of specific treatment.

Hansen reviewed the statistics of twelve authors who made reports between 1909 and 1939 on the bacteriology of acute suppurations of the middle ear. From the table which he presents, it is evident that the highest percentage of pneumococci, as against other bacteria, was reported by Wirth, of Heidelberg, Germany, in 1928 (39.9); the lowest percentage in any series was 8, reported by Jones, of New York, in 1935, and the average percentage in the entire twelve series was 21. Of these pneumococcic infections, 55 per cent were due to Pneumococcus type III, as against 45 per cent due to all other types of pneumococci.

Hansen reports on the bacteriologic findings in 182 cases of acute suppuration encountered at the Sundby Hospital, of Copenhagen, Denmark, between Dec. 1, 1938 and Sept. 15, 1939. In this series there were 48 cases of acute suppuration of the middle ear due to pneumococci. In 11 of these 48 cases infection was caused by Pneumococcus type I, in 13 by Pneumococcus type III and in 7 by Pneumococcus type XIX. In the remainder it was due to pneumococci of other types. Hansen states that if pneumococcic otitis is regarded as a whole, it is a relatively mild infection.

Of the entire group of 48 patients with pneumococcic infections, 22.5 per cent came to operation, as against 41 per cent of those with suppuration due to hemolytic streptococcus. Excluding the infections due to pneumococci of types III and XIX, which are most apt to give complications, the percentage of those due to pneumococci of the remaining nine types in which operation was done was only 10.5, while the operative percentage for the entire 182 cases was 23.2.

Together with other authors (Richter, Honda, Alberti and Wirth) Hansen speaks of pneumococcic otitis as a typical otitis of small children. While he notes that acute otitis in general is primarily a disease attacking small children (under 5), he does not find any particular difference in the incidence in patients of this age of pneumococcic otitis and that due to other organisms. He summarizes his reports of cases in which suppuration was caused by pneumococci of the various types substantially as follows:

Type I (11 cases). In only 1 of these was a mastoid operation necessary, and there were no complications in any of them.

Type III (15 cases). The classic picture of this variety is a sluggish, almost symptom-free development, with the eventual appearance of a free period, often prior to the development of an intracranial complication.

[Previous experience indicates that the type III pneumococcus is an organism which infects adults essentially, particularly elderly people, and rarely occurs in children. Hansen's 15 cases contradict this, inasmuch as 10 of the patients were children under the age of 5, while 3 were over 50. In these 15 cases the classic picture was encountered only three times, and always in patients over 40. In 2 cases the infection developed violently, and in the remaining 10 it developed in much the same fashion as a streptococcic otitis. All of the cases in which the infection developed in the ordinary manner were uncomplicated. This statement is in agreement with that of Maybaum and Druss that the type III infection is less virulent in children than in adults.—W. E. G.]

Type XIX (10 cases). In half of the patients ordinary otitis developed without complications. Of the others, 3, all under 1 year of age, had to be operated on after an aural discharge of seven to eight weeks' duration, and these patients showed considerable necrosis in the mastoid process. From the statistics it would appear that this type of infection also involves small children and is as dangerous as is type III in adults.

The otitides caused by the remaining types of pneumococci (V, VI, XI, XII, XIV, XV, XVIII, XXIII and XXIX) are quite innocuous. One exception was the case of a 1 year old child in whom meningitis with sepsis developed after acute type VI otitis, pneumococci of the same type being found in the spinal fluid as in the blood stream. The child died in spite of large doses of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and pneumococcic serum.

Meningitis developed in another child, one with a type XI infection. There were 4,800 cells in the spinal fluid. This child recovered after an operation and intraspinal administration of sulfanilamide.

GROVE, Milwaukee.

Pharynx

SIGNIFICANCE OF THE TONSILS IN THE DEVELOPMENT OF THE CHILD. A. D. KAISER, J. A. M. A. **115**:1151 (Oct. 5) 1940.

Kaiser discusses the significance of the condition of the tonsils in the development of the child by analyzing the tonsil problem as found in his thorough investigations as well as by commenting on the conclusions of other observers.

He admits the beneficial results attained in numerous selected cases and recognizes the value of surgical intervention as a prophylactic as well as a therapeutic measure.

Studies were made especially on tonsillitis, otitis media, common cold and cervical adenitis, and factors of development of the child were always considered in these studies. The conclusions drawn may be summarized as follows:

1. Marked hypertrophy of the tonsils, as well as repeated inflammations, giving rise to attacks of tonsillitis and cervical adenitis is frequently associated with impaired physical development, and tonsils thus affected should be removed after the age of 4, with the expectation that 50 per cent of the children operated on will be materially benefited.

2. The common cold, otitis media, sinusitis and laryngitis may influence the child's development unfavorably. The tonsils cannot be proved to be the causative factor; hence, tonsillectomy does not offer a solution for their eradication save in exceptional cases.

3. It could not be shown that the incidence of such diseases as bronchitis, pneumonia and tuberculosis was reduced in tonsillectomized children.

4. Rheumatic disease and nephritis constitute serious handicaps to a child's normal development, but recent studies seem to show that the tonsils and their removal play a less significant role in the causation and treatment of these diseases, respectively, than was formerly supposed. There has been a reduction in the number of throat infections following tonsillectomy, with benefit to rheumatic patients.

5. There has been a decline in the rate of mortality and morbidity among children during the last three decades as well as a definite improvement in the physical development of children in America. When careful consideration is given to the application of new discoveries in the field of nutrition, immunology and sanitation, it can be said that the tonsils are not as great a menace to a child as has been frequently suggested.

6. There is substantial evidence that in about 20 per cent of children the tonsils are either hypertrophied or diseased and that these conditions have an unfavorable influence on the development of the children. Such tonsils should be removed.

GORDON, Philadelphia.

THE TREATMENT OF BLACK HAIRY TONGUE (LINGUA NIGRA). J. WALKER TOMB, J. Trop. Med. **43**:155 (June 1) 1940.

Tomb defines black hairy tongue as "a rare condition in man due to the growth in the superficial tissues of the tongue of the fungus (Blastomyces) Cryptococcus vel Saccharomyces linguae pilosae. The false yeasts (Cryptococci) reproduce by budding and do not form mycelia or ascospores. Cryptococci (asporogenous yeasts) result when monilas lose their power to form mycelia. Rhizopus niger and Nocardia lingualis have also been found associated with this condition."

Histologic examination of the hairs shows them to consist of a central core of nucleated epithelial cells surrounded by a zone of acellular keratin-like material. The prevailing view as to causation is that there is a primary hyperkeratosis of the tongue, associated with hypertrophy of the filiform papillae.

Tomb describes a case of black tongue of four years' duration in a man in whom the condition had developed in India. The disease had been treated unsuccessfully with potassium iodide given internally as well as by various local applications. As an experiment it was decided to administer potassium chlorate internally (5 grains [0.3 Gm.] three times a day). By the end of ten days the tongue was apparently normal, and there was no recurrence eighteen months after the treatment. LAYMON, Minneapolis. [ARCH. DERMAT. & SYPH.]

TONSILLITIS TREATED WITH BISMUTH. G. MARASCHINI, Med. ital. **21**:65 (Feb.) 1940.

Maraschini reviews the literature of the past six years on the use of bismuth compounds in the therapy of tonsillitis. From the use of bismuth compounds in 44 of his own cases he concludes that bismuth is effective in the treatment of acute tonsillitis. There is a marked improvement in all symptoms in from twelve to twenty-four hours. Treatment is harmless if carefully carried out. A fat-soluble preparation of bismuth is used. The dose, ranging from 1.5 to 3.5 cg, is determined according to age. MARTOCCIO, Utica, N. Y. [AM. J. DIS. CHILD.]

Nose

ROENTGEN THERAPY FOR ACUTE SINUSITIS. HENRY L. WILLIAMS and WALTER C. POPP, Ann. Otol., Rhin. & Laryng. **49**:749 (Sept.) 1940.

The most striking effect of roentgen therapy on acute sinusitis is the relief of pain and headache. The relief of pain is often accompanied by a noticeable increase in discharge, so that the effect of the therapy may be due to a lessening of the engorgement of the nasal mucosa. When therapy was instituted early, more good results were obtained, and the results seemed better in initial attacks than in recurrent attacks. M. V. MILLER, Philadelphia.

FIBROMAS OF THE NASOPHARYNX. FREDERICK A. FIGI, J. A. M. A. **115**:665 (Aug. 31) 1940.

Figi presents a complete and scholarly review of fibromas of the nasopharynx, with schemas, roentgenograms and histologic observations. He summarizes and concludes as follows:

"Sixty-three cases of juvenile basal fibroma of the nasopharynx observed at the Mayo Clinic were studied. Fifty-eight of the tumors occurred in male patients; five were present in female patients. The patients ranged in age from 10 to 31 years at the time of examination. The nasopharynx was involved in all the 45 cases studied in detail, the nasal fossae in 34. The antrum, sphenoid and other accessory sinuses were invaded in a number of instances. Removal of tissue from these tumors is always attended by profuse bleeding and in some more recent instances of the condition biopsy was not performed, the diagnosis being based on

the history, the age of the patient, the hardness of the tumor and the characteristic clinical picture. Surgical removal of these tumors involves considerable risk, and recurrences are frequent. Implants of radium and electrocoagulation supplemented with radium are the most effective forms of treatment in these cases. By means of electrocoagulation and insertion of radium a tumor of this type can at times be eradicated with a single application, but fewer complications are likely to be encountered if treatment is carried out in stages. There was no mortality in the series. All the patients who received complete therapy are now well except for a boy, 16 years of age, who is still under treatment at time of writing."

GORDON, Philadelphia.

CRANIAL AND INTRACRANIAL COMPLICATIONS OF ACUTE FRONTAL SINUSITIS. PAUL C. BUCY and W. TRACY HAVERFIELD, J. A. M. A. **115**:983 (Sept. 21) 1940.

Bucy and Haverfield report 3 cases of cranial, intracranial and combined infection secondary to acute infection of the frontal sinus which followed swimming and 1 case in which such infection followed trauma to a stye. All of the patients were operated on and recovered.

From these cases and a review of the literature, certain conclusions were drawn, substantially as follows: 1. The infection usually spreads to the frontal bone and invades the brain by way of the venous system.

2. Prevention of the fulminating frontal sinusitis, osteomyelitis and brain abscess, which so often follow swimming, can in a measure be attained by instruction in proper breathing and the use of protective devices, such as nose clips or ear plugs, and by the avoidance of chilling. In acute fulminating disease, it is of the utmost importance to prevent the spread of infection by external surgical drainage.

3. Osteomyelitis should be treated by excision of all of the infected bone as soon as the diagnosis is made. One should bear in mind McKenzie's original dictum that "once progressive osteomyelitis has set in, the only chance of saving the patient lies in the immediate and entire removal of the diseased bone." This view apparently is now accepted by practically all neurologic surgeons and most rhinologists.

4. Brain abscess should be drained through an uninfected area of the skull, if possible, with due attention to prevention of infection of the meninges.

5. Defects made by removing infected bone from the skull are probably usually repaired by the spontaneous regeneration of bone.

GORDON, Philadelphia.

SINOFACIAL WOUNDS IN WAR SURGERY. M. ESCAT, Oto-rhino-laryng. internat. **24**:241 (Sept.) 1940.

Sinofacial wounds are wounds of the face which are complicated by open fracture of the walls of the pneumatic cavities. Owing to the particular anatomic relations, these wounds permit the use of intranasal drainage and complete closure of the external lesion. War wounds being considered, a priori, as highly septic, ample drainage appears to be the sole rational therapeutic indication; suture of the wounds is regarded by the majority of surgeons as warranted only under restricted conditions. Except for the region under discussion, suture and drainage of war wounds are irreconcilable. In sinofacial wounds ample drainage is not incompatible with hermetic suture of the integument. This applies to all such wounds, whatever the period which has elapsed since reception, whatever the state of infection or whatever the associated ocular or dental complications may be.

After the induction of appropriate anesthesia, the wound is carefully cleansed, every possible piece of integument being preserved. The cavity of the sinus is then exposed and explored, and after the removal of foreign elements and denuded bone particles a large counteropening is made into the nasal cavity and gauze

drainage is inserted. Postoperative care is simple. The external dressing is dispensed with after the third day, the drain is removed in twenty-four to forty-eight hours, and if the nature and abundance of the secretions demand it, lavage of the sinus is done—but not until after five days have elapsed. The skin sutures are removed on the fifth day.

Six illustrative observations are recorded. DENNIS, San Diego, Calif.

Miscellaneous

ATYPICAL FACIAL NEURALGIA. MARK ALBERT GLASER, Arch. Int. Med. **65**:340 (Feb.) 1940.

Glaser believes that there is a type of facial neuralgia transitional between migraine and trigeminal neuralgia, which he designates as primary atypical facial neuralgia. It is characterized by pain, which is always deep seated and aching, burning or throbbing in character. No specific factor could be held responsible for the onset of the pain, which in 33 per cent of the cases was bilateral. The distribution of pain was in a circular area within the distribution of the vascular supply of the face and head. It is frequently aggravated by such local factors as cold, heat, eating, worry, constipation and exertion. In 50 per cent of the cases of atypical neuralgia, sympathetic phenomena, such as lacrimation, blurred vision, unequal pupils, photophobia and corneal infection, are present. The patients, who are chiefly females, have continual annoying pain with superimposed severe exacerbations. There is an associated neurosis, which is believed to develop secondarily to the persistent annoying, irritating pain. Addiction to narcotics is not uncommon in association with atypical neuralgia, while it is rare with trigeminal neuralgia.

The author gives the following useful classification of atypical facial neuralgia.

I. Primary atypical facial neuralgia

II. Secondary atypial facial neuralgia due to various clinical entities
   1. Sphenopalatine and vidian neuralgia.
   2. Postherpetic trigeminal neuralgia
   3. "Trigeminal ghosts"
   4. "Trigeminal ghosts" with lingual spasm
   5. Syndrome due to abnormalities of the mandibular joint
   6. Autonomic faciocephalalgia
   7. Painful convulsive tic
   8. Headache due to hypertonicity of muscles of the neck
   9. Senile neuralgia

III. Atypical facial neuralgia produced by systemic diseases
   1. Allergy
   2. Endocrine disturbance
   3. Psychoneurosis

IV. Atypical facial neuralgia due to lesions of the head, chest and abdomen
   1. Infections about the head
      (*a*) Mastoiditis
      (*b*) Thrombosis of the cavernous and longitudinal sinuses
      (*c*) Deep-seated facial abscess
   2. Tumors of the head and neck
   3. Intracranial lesions
   4. Dental sepsis
   5. Deviations and spurs of the nasal septum

6. Ocular lesions
7. Lesions of the chest
8. Pathologic conditions in the abdomen and pelvis

The pain of atypical neuralgia is of a burning, pressing, aching, discomforting nature. It is not acutely lancinating, as in peripheral nerve neuralgia. The atypical neuralgia is not identical with the distribution of any cranial nerve, but does follow to a degree the course of the vasculature of the head.

In the treatment of atypical facial neuralgia every effort should be made to eliminate the secondary factors which may precipitate the pain. The best results are obtained in those cases in which the pain has been of short duration. Medical treatment consists of the use of the choline compounds, as well as epinephrine, ergotoxine, amyl nitrite, physostigmine and atropine. Resection or blocking the cervical sympathetic fibers has afforded relief in some cases, but it is strongly recommended that all medical measures be employed and exhausted before any operative procedure is carried out.

BECK, Buffalo. [ARCH. NEUROL. & PSYCHIAT.]

MANAGEMENT OF FACIAL PARALYSIS BY PHYSICAL MEASURES. M. K. NEWMAN, J. M. BERRIS and S. S. BOHN, Arch. Phys. Therapy **21**:270 (May) 1940.

Newman and his co-workers state that the treatment of paralysis of the facial nerve is dependent on its various etiologic factors, on the question of whether the paralysis is of central or of peripheral origin and on the site of the lesion. They managed 30 patients by the use of physical therapeutic measures. The ages of the patients ranged between 6 and 79 years, the average age being 37. With the onset of an acute refrigerans type of facial paralysis, iodides in saturated solution of potassium iodide or syrup of hydriodic acid are indicated for their general alterative action. Analgesics are valuable. Saline laxatives are used during the acute stage of inflammation, for their possible detergent effect. The physical measures applicable during the acute phase of the refrigerant palsy are directed toward the reduction of the inflammatory exudate and toward keeping the denervated muscles in a position of physiologic rest. Decongestion at the stylomastoid foramen can be accomplished by cantharides plasters, hot moist applications, radiant heat, negative galvanism and conventional or short wave diathermy. The frequency of application is indicated by the pain and the tolerance of the tissues. To prevent muscles from stretching, they are supported by attaching two Y bands of adhesive tape at the zygomatic bone and temporal region. Positive galvanism is especially efficacious for the relief of pain. With the subsidence of the acute symptoms within eight to ten days, testing for reaction of degeneration is instituted. When no reaction of degeneration is present, recovery can be expected in from two to eight weeks. If a sufficient amount of recovery is not obtained after six months of rigorous physical therapeutic measures, surgical intervention is to be considered. Active treatment now consists of heat, massage, voluntary exercise and electrical stimulation (with interrupted galvanic, sinusoidal and faradic currents). Diathermic or radiant heat is applied to the paralyzed side of the face for from fifteen to thirty minutes. Another method of promoting a circulatory response consists of the common ion transfer of 0.5 per cent acetylbetamethylcholine hydrochloride. From 15 to 20 milliamperes of the galvanic current for twenty minutes is adequate. Negative galvanism, for its heating effect, can be applied for thirty minutes over the stylomastoid foramen. The strength of current should be just less than the amount required to cause a contraction of the muscles on the unaffected side. On the first day three contractions over the motor points are sufficient, and on subsequent days the number of contractions is increased but always kept within the fatigue range of the muscle. After one or two weeks the slow sine wave current is substituted, from twenty to thirty contractions per minute being used. When the faradic current can be tolerated, it should replace the sine wave current. Gentle stroking massage, which follows stimulation, pre-

serves muscular tone. Exercise of the facial muscles is carried out in front of a mirror every day. The causative agent must always be removed before physical measures are instituted. In 17 of the 30 cases the paralysis followed exposure; in 6, disease of the middle ear; in 1, dental manipulation; in 1, trauma associated with an automobile accident; in 2, head colds; in 1, mumps; in 1, an operation on the eighth nerve for vertigo; in 1, postpartum infection, and in 2, mastoidectomy. In the 17 cases of paralysis due to exposure, partial or no reaction of degeneration was present after ten days. Eight of these patients received only heat, massage and exercise, while the remainder received electrical stimulation as well.

J. A. M. A.

THE UNSOLVED PROBLEMS IN APHASIA: III. AMNESIC APHASIA. J. M. NIELSEN, Bull. Los Angeles Neurol. Soc. **5**:78 (April) 1940.

The patient with amnesic aphasia is alert, cooperative and able to talk and explain that he "knows but can't say it." His concepts are clear, except for their verbal symbols, which he cannot recall. In some cases only nouns, in others verbs, are lacking. The words cannot be recalled spontaneously or after visual or tactile perception of the object but can be recognized when heard or seen. In transcortical motor aphasia, on the other hand, the patient does not have a clear idea of the word he wishes, repeats indiscriminately any word offered him, has difficulty with all parts of speech and talks little. The location of the lesion evoking amnesic aphasia is in doubt, having been placed variously from the frontal to the posterior part of the temporal lobe. This type of aphasia unquestionably may arise from lesions of the posteroinferior portion of the major temporal lobe. It is found in some cases of lesion of the angular gyrus, but not in all. Nielsen briefly describes 7 cases from the literature which indicate that amnesic aphasia may be due to lesions involving the supramarginal gyrus in the major parietal lobe. In all these cases impingement on the posterior portion of the superior temporal convolution was immediate. In almost all cases there was disturbance also of writing and reading. Nielsen concludes that amnesic aphasia results from a lesion of the major posterior temporo-occipital region or its superior connection with the rest of the brain through the temporal isthmus—Brodmann's area 37 and the posterior portions of areas 21 and 22.

MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

HEREDOPATHIC COMBINATION OF A CONGENITAL DEFORMITY OF THE NOSE AND OF MYOCLONIC TORSION DYSTONIA. L. BENEDEK and E. RAKONITZ, J. Nerv. & Ment. Dis. **91**:608 (May) 1940.

Benedek and Rakonitz report the heredofamilial occurrence of combined developmental retardation of the nasal bones and a form of myoclonus and torsion dystonia in three generations of the same family. A mother and her 2 sons, all affected, were examined; her maternal aunt was known to have had the same abnormalities. The nasal anomaly consisted of a median nasal groove, similar to that seen in certain anthropoid apes (Simia satyrus L.), with hypoplasia of the minor and major alar, the septal and the lateral cartilages. The motor disturbances consisted of rapid, spontaneous, arrhythmic, involuntary contractions of the broad flat muscles of the shoulders, neck and back, without resulting motion of the extremities (myoclonus) and, in addition, torsion spasm of the muscles of the neck and trunk. The patients examined also exhibited enlargement of the accessory nasal and mastoid cavities and infantilism and sclerotization of the larynx. The nasal and laryngeal abnormalities were equally manifest in all the affected persons, but the intensity of the motor disturbances varied from one patient to another. Emotional factors influenced both the myoclonus and the torsion dystonia. Benedek and Rakonitz consider the whole complex heredopathy to be due to a developmental disturbance of the mesencephalon and diencephalon during the first three or four months of embryonic growth.

MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

AN EXAMINATION OF THE OLFACTORY BULBS IN FATAL CASES OF POLIOMYELITIS DURING THE VICTORIAN EPIDEMIC OF 1937-1938. E. G. ROBERTSON, M. J. Australia **1**:156 (Feb. 3) 1940.

A study of 20 olfactory bulbs, 9 pairs and 2 single specimens, from 11 patients who had died of poliomyelitis afforded no definite evidence for or against invasion of the nervous system by the olfactory pathway. In 1 olfactory bulb from a series of 9 patients with early pharyngeal or respiratory palsy or both, inflammatory changes were noted. In 3 olfactory bulbs from 2 patients with paralysis of the skeletal muscles and without pharyngeal paralysis, no inflammatory changes were noted. The accumulated evidence suggests that the virus may reach the central nervous system by pathways other than the olfactory and that when bulbar palsy is an early manifestation the afferent pathway is probably along the glossopharyngeal nerves from the pharyngeal and tonsillar regions.

GONCE, Madison, Wis. [AM. J. DIS. CHILD.]

INFLAMMATORY CONDITIONS OF THE UPPER PORTION OF THE RESPIRATORY TRACT. R. A. R. GREEN, M. J. Australia **1**:179 (Feb. 10) 1940.

In infection of the upper respiratory tract the lymphoid tissue in the nasopharynx is the most important factor to consider. In addition to the arrest of infection, this tissue probably aids in the development of immunity.

In the maintenance of the normal anatomic structure of the nose, methods of feeding are considered important. The work involved in breast feeding tends to produce a lower palatal arch and a roomier nose.

The common cold must always be regarded as a possible forerunner of serious disease. Rest in bed and treatment of the nose until drainage has ceased are important.

In chronic disease of the sinuses, infected tonsils play an important role. It is not unlikely that unhealthy conditions in the nose and nasopharynx favor attacks of other diseases, such as poliomyelitis.

The abundant sunlight and available good diet of Australia should make these infections less numerous if these factors are of importance. It is felt that a considerable number of the patients who require hospitalization do so on account of such infections and that there is the possibility that the coastal position, with moist atmosphere, may be one reason for their prevalence.

Adenoids should be removed whenever they are found enlarged, with frequent colds or disease of the middle ear. Unless they are grossly infected, tonsils should be left until the age of 5 years.

TENNEY, Madison, Wis. [AM. J. DIS. CHILD.]

TRUE CONGENITAL FACIAL PARALYSIS. ATILIO VIALE DEL CARRIL, Rev. argent. de oto-rino-laring. **9**:85 (March-April) 1940.

After a review of the literature del Carril presents a case of true congenital facial paralysis. Assuming that the etiologic factors in this patient could not be altered by any type of treatment, he employed symptomatic measures to correct the deformity.

He noted that on account of atrophy of the facial muscles there was a deviation of the lower jaw to the healthy side and that, in consequence, there was a definite malocclusion of the teeth. He made an orthodontic prosthesis, which corrected this malocclusion. In addition, the patient was instructed to take daily muscular exercises to strengthen the facial muscles.

After a year and a half of observation, he found that the general appearance of the patient was markedly improved, and his photographs show the patient "before and after" treatment.

PERSKY, Philadelphia.

Society Transactions

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GORDON BERRY, M.D., *President*

WILLIAM E. GROVE, M.D., *Editor of Abstracts*

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Practical Technic for Tracheotomy. DR. FREDERICK T. HILL, Waterville, Maine.

Laryngeal obstruction is the one outstanding emergency calling for the services of the laryngologist. Often tracheotomy must be performed under unfavorable conditions and the patient left with physicians who are inexperienced in the after-care. Certain modifications in technic, none of them original, have been adapted into a routine for such patients. The objective is to convert an emergency into an orderly procedure by eliminating the immediate danger of asphyxia and to allow for an easier and safer after-care by inexperienced personnel. The main points are the intubation of the trachea with a bronchoscope and the use of the Mosher "punch tracheotomy" technic.

DISCUSSION

DR. HARRIS P. MOSHER, Marblehead, Mass.: I am much obliged to Dr. Hill for getting this technic into print systematically. I have discussed it a number of times, but I never have written it up. It is now in systematic form and goes into print for whatever it is worth.

The original life saver was the bronchoscope, and it has advantages over the modifications, but the modified tube has its place for emergency intubation. For the routine tracheotomy it is not as good as the small bronchoscope.

It is unusual—and I think it sounds egotistical—to have to go to somebody else to get the right name for a procedure that one has proposed. In "window" technic, however, Dr. Hill has given it a much better name than "punch tracheotomy," which does suggest an undue amount of force.

The chief advantage of this procedure is, as Dr. Hill pointed out, the safety which it brings to the patient when he leaves the hospital.

When the old method with the tube was used the first night, even in the hospital, was often an unpleasant affair. This procedure provides safety when the operation has to be done outside the hospital and the patient left in inexperienced hands. That, to me, is its biggest advantage.

Laryngologists who work in hospitals, with every convenience about them, do not realize the anxiety of the operator when he has to do a tracheotomy where the equipment is not complete for his convenience and for the safety of the patient.

DR. CHARLES T. PORTER, Boston: There are one or two points that Dr. Hill mentioned briefly which I should like to emphasize a little more.

There is no question but that the punch tracheotomy is a procedure with which the laryngologist can make an opening as quickly as possible. It takes out all the tissues between the exterior and the lumen. It is so named because the ring points on the back of the basket are used to take out the edges of the trachea.

It is important that one guard against the loss of tabs of tracheal rings by using the punch with the basket on it or else by using a small knife and taking hold of the ring with a forceps. However, I do know of cases in which pieces of the ring have been lost in the trachea and it has been necessary to search for them farther down. That increases the length of the operation a bit and does not help the patient.

There is one other point, having to do with stenosis. I saw this occur in a patient whom I sent to the Eye and Ear Infirmary last year. With small children one must be especially careful not to take out too much of the tracheal ring, because if one does, partial stenosis occurs after the wound is healed and may last a considerable time. I have seen 2 children with stenosis. In adults, the lumen of the trachea is large enough to allow for a good deal of leeway.

One other point, I think, is important, and that is the changing of the tracheotomy tube. I have a patient in my service now, who was transferred to me, on whom the operation was done through the surgical service in another hospital. The tracheotomy tube was not changed often enough or did not fit well enough, and there is present a tracheoesophageal fistula. If a tube remains in for a long time there is the possibility of necrosis.

Dr. Thomas C. Galloway, Evanston, Ill.: This may be a timely presentation, because there is a circumstance, if one may judge by the published reports, in which wholesale use of tracheotomy may be necessary, and that is the bombing of civilian populations with the liberation of pulmonary irritant gases, for example, phosgene. The reports would indicate that, in addition to pulmonary edema, there is a type of bronchitis much like that seen after a flame injury, with accumulation of secretion, secondary vascular changes, necrosis, etc., in the treatment of which tracheotomy may be life saving.

Animal experimentation to prove this point is now being undertaken, and it is obvious that if the premise is correct this type of operation may be extremely valuable.

It is interesting to note that in the laboratories of the department of physiology at Northwestern University, where it is extremely difficult to keep a dog alive after tracheotomy, a procedure of this sort, modified from Dr. Mosher's procedure, has been used for some time in tracheotomies on dogs. The animals do not need after-care with punch tracheotomy. They all die if the tracheotomy tube is left in.

I should like to know if it is not possible to do a true punch tracheotomy in which a cutting element is introduced along with the trocar of a mechanical tracheotomy and in which a tapered tube is used to control bleeding.

Dr. Gordon Berry, Worcester, Mass: I will ask Dr. Hill to close the discussion, and in closing it, will Dr. Hill say something about whether he takes out any ring tissue? I do not think that was clearly brought out.

Dr. Frederick T. Hill, Waterville, Maine: Answering Dr. Berry's question first. Yes, there is a portion of one tracheal ring removed with the flaps which are taken out in this crucial incision.

I think Dr. Galloway's bringing up the possibility of new conditions incident to war is interesting. Physicians and surgeons will probably have to readjust themselves a great deal. It will be worth while to be able to simplify procedures which may have to be done under a great number of adverse circumstances.

Dr. Porter's suggestion about the size of the opening is good. I try to make this opening to fit the proper tube, trimming it so that the tube will not fit too tightly but, of course, not removing an excessive amount of tracheal tissue.

I want to thank Dr. Mosher for the original subject, for the opportunity of getting helpful instruction from him many years ago and for his kindly discussion. Especially, I should like to thank him for his agreeing to the renaming of the procedure.

Lymphoid Tissue in the Upper Respiratory Tract. Dr. D. C. Jarvis, Barre, Vt.

This paper presents a number of clinical observations made during a twelve year study of the behavior of lymphoid tissue in the upper respiratory tract. These observations include the following ones:

1. A relation exists between the intake of iodine and the amount of lymphoid tissue in the upper respiratory tract.

2. A relation exists between the law of halogen displacement and lymphoid tissue in the upper respiratory tract.

3. Calcium in excess opposes the hypertrophy of lymphoid tissue in the upper respiratory tract.

4. Phosphorus in excess produces hypertrophy of lymphoid tissue in the upper respiratory tract.

5. From clinical results, iodine appears to be antagonistic to phosphorus.

6. The significance of the presence of lymphoid tissue in the upper respiratory tract is the guide it furnishes to the status of calcium and phosphorus metabolism at the time of examination.

7. By influencing calcium and phosphorus metabolism, especially phosphorus metabolism, one is able to influence the size of lymphoid tissue in the upper respiratory tract.

DISCUSSION

DR. BURT R. SHURLY, Detroit: This paper is of great significance and extreme importance and is a key to some of the problems of metabolism that specialists are perhaps prone to forget in concentrating their observations directly on the throat itself.

For many years, under the wonderful training that I received from Dr. E. L. Shurly, I found the problems of internal medicine as connected with the throat extremely interesting.

The study of lymphoid tissue, so long a mystery to the medical profession, is, of course, a study of the greatest importance.

Iodine content, for which a special blood test has been developed, is an important problem, and the iodine content is undoubtedly one of the balancing ingredients of the human system.

In Michigan many years ago a survey was made on thyroid disease and thyroid hypertrophy, and in the schools of the five lower counties it was found that 53 per cent of the children showed some thyroid disease. Of course, one must remember that Michigan is an area of thyroid disease almost the equal of Switzerland. That the glacial drift came down and took the iodine content out of the soil is a good theoretic explanation. However, iodized salt was introduced in the restaurants where 50,000 children were fed each day, and in a second survey, made about seven years later, it was found that only 7.2 per cent had thyroid hypertrophy.

What this may mean, of course, is that salt containing iodine is of extreme value in the diet, that a small amount of iodine is sufficient to help establish a balance of the metabolism and, certainly, that iodine affects the thyroid gland, which is a stabilizer of many elements in the human system. The manifestations in the throat of a lack of thyroid or an increase of thyroid are familiar.

The interesting thing to me is that for many years, without good scientific knowledge but with good clinical observation, children with malnutrition, many of them tuberculous, were given a prescription of malt and pulverized bone which has a high content of calcium, dilute phosphoric acid and lactic acid. Compound solution of iodine U. S. P. was also given. This balance of mineral salts and vitamins, malt and bone and phosphoric acid, allows children who do not thrive after tonsillectomy but remain puny, weak and anemic to receive a marvelous stimulation.

I believe that laryngologists must make a greater study of diet and of metabolism in general and that many patients with a lack of calcium or an increase in calcium can be definitely aided. The amount of calcium can be determined by means of a blood test. Laryngologists should make more extensive use of laboratory methods. After the amount of iodine in the blood has been determined, all the remedies are at hand to increase or decrease the amount of mineral salts in the blood stream.

DR. FRANK R. SPENCER, Boulder, Colo.: It is agreed that the importance of biochemistry is increasing. Dr. Mosher said a few years ago that it will solve more and more of our problems in the future. I occasionally see adults who present less of a problem than children.

I have been using Dr. Lillie's remedy, lipoiodine tablets, for lymphoid tissue in the pharynx, and these have given good results in patients who are not very ill.

I have Dr. Crowe's applicator, which he described before the society a year ago, in Rye, N. Y. I have under my care a student from the Mississippi Valley who has recurrent attacks of infection of the lymphoid tissue of the nasopharynx. Soon I am going to have him treated with radon seeds in order that he may not go through another winter with so many acute attacks. The fact that this radon applicator can be placed in the nasopharynx and treatment be completed in about two minutes on each side constitutes an advantage over treatment with roentgen rays.

Dr. Jarvis has previously discussed radium therapy of the nasopharynx and has said that it destroys many mucous glands and sometimes is too radical for lymphoid tissue. The radon applicator offers an opportunity for treatment which is not radical but brings good results. According to Dr. Crowe, the treatment may be repeated from time to time in order to accomplish further results if the first one or two treatments have been inadequate. This is not always possible with roentgen rays.

DR. T. E. CARMODY, Denver, Colo.: Dr. Jarvis has given some new thoughts, especially on calcium. Most treatment for lymphoid tissue has been, as Dr. Jarvis said, empiric:

I have found that many patients, especially after the tonsils have been removed, have lymphoid tissue, most often at the base of the tongue, at the lingual tonsil, and that it often causes them trouble in trying to clear the throat, particularly when they lie down at night.

For a number of years I have found the best remedy to be 5 to 10 drops of compound solution of iodine U. S. P. in a quarter of a glass of water or milk.

In connection with Dr. Jarvis' remarks about fats, many people who suffer from cold hands and feet may need more fat, but many will do well with small doses of thyroid; possibly they should receive fats also.

Dr. Shurly called attention many years ago to the relation of infected tonsils to the thyroid, and I think it is possible that in some cases the tonsils are removed too early.

In place of compound solution of iodine U. S. P., one can use hydriodic acid or Amend's solution, of which Dr. Jarvis spoke.

Dr. Spencer spoke of radon treatment as described by Dr. Crowe last year. I think that this treatment is overrated, and I think the use of roentgen rays is also overrated. Of course, laryngologists who have radium can use it, but the radon seeds are very short in application. With radon seeds one may do something to the lymphoid tissue, but, as Dr. Spencer said, one may also do something to the glands and the other tissues and probably do a great deal of harm.

Iodine, of course, has been used from time immemorial, and I am glad to hear Dr. Jarvis come out with the statement that it is of considerable help.

DR. GEORGE B. WOOD, Philadelphia: As I grow older, I find that I am getting increasingly conservative as far as radical removal of the tonsils is concerned, though of course I still recognize the importance of tonsillectomy and adenoidectomy when the proper indications exist. Especially is conservatism to be practiced in the management of tonsillar conditions in early childhood, when the removal of large tonsils is apt to be followed by compensatory hypertrophy of the other lymphoepithelial tissues in the throat. If, therefore, one can find a medicinal way of reducing the lymphoepithelial tissue in the throat in early childhood, one will have added an important item to therapeusis. As I understood Dr. Javis, he said that fat in the intestinal tract absorbs iodine from the food and carries it into

the system and that therefore a diet rich in fat would increase the iodine in the body and thus tend to reduce the amount of lymphoid hyperplasia. Am I right?

DR. D. C. JARVIS, Barre, Vt.: Yes.

DR. GEORGE B. WOOD, Philadelphia: In discussing adenoid tissue of the nasopharynx before this association a year ago I called attention to the work done by Dr. Clark, professor of anatomy, at the University of Pennsylvania in 1923 (Clark, E. R.: Studies on the Relation of the Size of Palatine and Pharyngeal Tonsils to Diet, *Tr. Am. Laryng., Rhin. & Otol. Soc.* **30**:438, 1924). This was an elaborate study of the influence of diet on the increase of tonsillar tissue in kittens. I had personally talked with Dr. Clark about this work and had gone over his papers and illustrations. It seemed to me that his results were very factual and that there was no doubt that a high fat diet and to a less extent a high protein diet increase markedly the size of the tonsillar tissues and also that of Peyer's patches and other lymphoepithelial structures of the gastrointestinal tract. These results suggest that a fat-free diet is indicated in tonsillar hyperplasia, which is contrary to Dr. Jarvis' suggestion concerning the importance of fat as a vehicle for introducing iodine into the system.

DR. HOWARD C. BALLENGER, Winnetka, Ill.: One point has not been touched on—and I feel a little bit worried about it—and that is the question of heredity. To me heredity has been the most important factor, or, rather, I assume it has been the most important factor, in lymphoid hyperplasia. Perhaps Dr. Jarvis has the answer in the tendency in calcium and phosphorus metabolism. That may be the factor which is inherited.

I should like to ask Dr. Jarvis whether, in his investigations, he used familial cases to determine whether there is any difference in the metabolism of the phosphorus and the calcium.

DR. D. C. JARVIS, Barre, Vt.: Discussion of some of the questions which have been brought up would require considerable time. Taking the last one, the matter of inheritance, I happen to practice medicine in a little city which has a mixture of some fourteen different nationalities, one third of the citizens being Italian and one third Scotch and the other twelve nationalities representing almost all of Europe and the British Isles.

I soon learned that the so-called Nordics—persons from Norway, Sweden, Denmark, France, Ireland, Scotland, Wales and England—constituted most of the patients with lymphoid hypertrophy. Nordic groups customarily have diets, determined by their ancestors, which are low in phosphorus. They acquire chronic fatigue easily. While the phosphorus intake is too low, the calcium intake is too high. Therefore, in children who spring from a Nordic racial stock one should watch the blood phosphorus level. They should be encouraged to use vinegar and other foodstuffs which will raise the blood phosphorus.

With regard to the influence of diets high in fats on the tonsillar tissue of kittens, I refer Dr. Wood to the work by Dr. Pottinger, who has done so much experimental feeding of cats with special diets. Dr. Pottinger apparently has, by this means, thrown cats into the edema zones. Asthma and other conditions have developed. I think that in the work of Dr. Pottinger Dr. Wood will find the answer he wishes.

With regard to cold hands and cold feet. If one ventures very far into the field of biochemistry, one will come to the conclusion sooner or later, I believe, that acid is the guardian of the peripheral vascular bed. Cold hands and feet seem to vanish if the patient is given acid, as vinegar, hydrochloric acid or cranberry juice, which is very acid, or in any other form desired. The administration of acid, of course, depends on how much of a deficiency there is. Acid helps a great deal in restoring the balance in the peripheral vascular bed.

One or two observations might be interesting. I said that Vermont has the highest death rate for heart disease in the United States. I find that certain things happen to cardiac conditions which make one suspect that the underlying condition in heart disease is of functional origin, such as edema of the heart muscle or other

edema connected with the heart. In a person in the sixties, for example, the heart skips every fourth beat. In two weeks it does not skip at all if the patient takes chlorophyll and minerals, all well represented in kelp tablets. He should take only 1 a day. If given 6 or 8 he will have a gastric upset in two weeks. The pain of angina pectoris disappears inside of two weeks with 1 tablet a day and stays away. I do not say the condition is cured, but the pain stops. I have seen a heart murmur disappear in two weeks when chlorophyll and these minerals were given. They raise the blood phosphorus if it is too low and bring it down if it is too high, but in this case they apparently control the edema.

Each environment is different. My own environment is rich in calcium. I have to fight this all the time by raising the blood phosphorus level.

The nose and the throat are going to be the biochemical windows to the body, and when otolaryngologists look on them as such they will see that a biochemical imbalance is behind some clinical conditions.

An Experimental and Clinical Study of the Common Cold. Dr. Irwin G. Spiesman (by invitation), Maywood, Ill.

Thermoelectric studies of the temperature of the mucous membrane as an index of vascular changes show that rapid cooling of most cutaneous surfaces produces reflex vasoconstriction and ischemia leading to lowered temperatures of the mucous membrane of the upper respiratory tract.

The temperature of the nasal mucosa in the experiments conducted was measured with a thermopile applied to the membrane of the nose and the electromotive force measured through a potentiometer.

A normal response to a cold stimulus applied to the cutaneous surface is first a lowering of the temperature of the mucous membrane and next, with the stimulus still applied, a rise and general return to the initial temperature. Irregular reactions follow severe emotional activity and produce in otherwise normal persons abnormal reactions, such as a delayed reaction, no response whatever and a reversal of the normal response to a cold stimulus. With the manifestations of these irregular reactions this paper is particularly concerned.

DISCUSSION

Dr. Arthur W. Proetz, St. Louis: It seems to me that Dr. Spiesman's calling attention to the vasomotor influences in the production of the common cold is important. Sometimes I think that the vasomotor responses may be the effect as well as the cause of colds. The preliminary experiments which Dr. Spiesman described verify bit by bit those of Mudd, Goldman and Grant, made some time ago, and I merely want to emphasize one thing, that the reaction in the nasal mucosa lasts for an indefinite time after the cutaneous reaction has subsided. In other words, the relatively small and relatively transient chilling of the cutaneous surface caused by air conditioning may be accompanied with a much longer effect on the mucous membranes.

I do not quite understand the mechanism of the reversed reaction and what it means, because to my mind the mechanism of the cold in a person with reduced mucosal circulation is simply that the glands function in some subnormal manner, that drying takes place and that a ciliary insufficiency occurs, producing the cold. This leads me to the point I wanted to make: Colds can be controlled not only in the interval, as Dr. Spiesman suggested, but after they have ostensibly begun by simply attending to the vasomotor system and to the local disturbances which the aberrations of the vasomotor system have brought about. In other words, a patient feeling that he has a cold coming on has really not a cold as yet. He has prodromal symptoms which are not the results of infection by bacterium or virus but are the vasomotor reactions to which Dr. Spiesman referred. So if a patient, feeling a cold coming on, will within the first six or eight hours get his nose moistened and heated by the inhalation of steam, the cold can be aborted. To my mind the cold has not yet begun until the aberration of the cleansing mechanism of the nose is so pronounced that virus and bacterial penetration can take place.

DR. HERMAN M. TAYLOR, Jacksonville, Fla.: Dr. Spiesman has made clear the interrelation of the innervation of the peripheral blood vessels and those of the nasal mucous membrane. This autonomic control of vasoconstriction and vasodilatation forms the basis of the physiologic balance between the splanchnic and the peripheral nerves.

Thus, when stimulation by chilling results in peripheral ischemia, the nasal mucous membranes undergo a similar experience. Prolongation of this condition serves to throw out of balance the nicety of integration existing under normal metabolic conditions and accounts for the ability of ordinary virulent organisms to overcome the obstacle of tissue immunity at the portal of entry.

Dr. Spiesman has gone into the study of other phases, and I infer from what he says that he feels that lowered resistance, whether due to mental anxiety, worry or errors in diet, has more or less the same effect on the nasal mucosa.

Some time ago I reported an experiment with healthy, mature guinea pigs, weighing approximately 250 Gm. Each animal was in an ice refrigerator, the temperature of which was 15 C., for a thirty minute period, and immediately after removal a blood count was made. This showed leukopenia, with an average reduction of polymorphonuclears and neutrophils.

I also reported an observation on ten bathers who remained submerged for fifty minutes in water at a temperature of 22 C. They were kept quiet and refrained from all exercise. Blood counts were done, and all showed leukopenia with reduction in the polymorphonuclears and neutrophils. These observations were in contradistinction to the result of another experiment. Eight swimmers were subjected to exposure in ocean water at a temperature of 22 C. but had to swim for forty-five minutes. Immediately afterward blood counts were made, which showed leukocytosis, with an average of 10,000 white cells. It is evident from these observations that the reaction to chilling as a result of lack of muscular exercise is entirely different from the reaction to cold when the subject is carrying on muscular exertion. Five of the bathers were put into water at 22 C. for fifty-five minutes and were asked to refrain from taking any exercise. Of all the unhappy persons I have even seen, they were the most unhappy. They were all chilled. They refrained from any conversation. Finally they could not stay in the water any longer, whereas the active swimmer, who could stay in the water much longer, did not suffer such discomfort.

As I have said, leukopenia appears in the person who is chilled—in the swimmer who is inactive; and in the person taking a definite amount of muscular exercise just the opposite, leukocytosis, develops.

I think it would be interesting if Dr. Spiesman would also carry on some studies of the blood to determine whether there is leukopenia or leukocytosis.

As the result of the observations I made, it seemed logical to conclude that during exposure peripheral vasoconstriction, which also means ischemia of the nasal mucous membrane, is prevented by muscular activity, and it follows that infection is then less likely to occur. It follows also that the chilling of the body surfaces without compensatory exercise leads to morbid changes in the body. These changes cause vasomotor peripheral constriction, peripheral stasis, anoxemia, a lower leukocyte response, polymorphonuclear and neutrophilic impairment of phagocytosis and incapability of the fixed tissue cells, including those of the nasal mucous membranes.

One of the immediate results of these changes is the predisposition to infection of the upper part of the respiratory tract, the paranasal sinuses, the eustachian tubes and the middle ear.

DR. ERNEST M. SEYDELL, Wichita, Kan.: Last year I presented before this body an article on nodular spastic myalgia of the neck muscles. When persons with this condition expose themselves to a draft, they have an ischemia of the neck muscles from which headaches and other pains may develop.

I have felt that a certain percentage of persons are subject to this condition. I have never been able to trace the condition to worry.

I think it would be a good idea for Dr. Spiesman to note in his further experiments whether myalgia develops in persons who have delayed reactions.

DR. JAMES A. BABBITT, Philadelphia: In Philadelphia there is an allergist who is a very good allergist and likewise a very good internist, Dr. Richard Kerr. He had two or three patients who were suffering from some type of bronchial asthma, some type of bronchial blocking. He gave them the simple direction of closing their windows instead of opening them at night and not allowing their rooms to become cold while they were sleeping.

Many patients who frequently have colds will tell of having a cold air fad and of following the practice of breathing cold air to become resistant.

DR. IRWIN G. SPIESMAN, Maywood, Ill.: The question brought up by Dr. Proetz is one which has bothered laryngologists for some time; that is, why is there a reversed response to a cold stimulus and why is there a sluggish response physiologically?

Dr. Gelhorn, who is a vasomotor physiologist of considerable repute—and I have watched his work and talked with him a great many times about this—has no real explanation except to say, as Cannon has shown, that emotions affect the sympathetic-adrenal system, first, of course, directly affecting the vasomotor centers, then acting through the sympathetic chain, and then working directly through the adrenals. Thereafter the various changes occur.

Gelhorn said that when an emotion upsets the subject or when an emotional state is present, not only the sympathetic-adrenal system but the parasympathetic system is stimulated.

Cannon expressed the belief that when the parasympathetic response developed it was of pathologic significance. Gelhorn stated the opinion that it is a normal occurrence. He further stated that the parasympathetic response may overshadow the sympathetic response in some conditions. Normally, the sympathetic response overshadows the parasympathetic.

Fright is an example of the parasympathetic action overshadowing the sympathetic. In fright vasodilatation appears in many organs. If there is a reversal of the normal reaction, if the cold stimulus applied now to the skin sends the temperature up instead of down, there will probably be a parasympathetic response to a cold stimulus provoked by fright.

Normally, the temperature of the nasal mucous membrane responds to cold by lowering the temperature in the nose. In fright, as has been demonstrated in several cases, there is an increase in the nasal temperature. That is, the parasympathetic system, being most active, will increase the blood supply in the nose, and the nasal temperature will rise.

Next, the reason for the delayed reaction is not entirely clear from a physiologic standpoint. However, chronic worries, anxieties and fears affect the sympathetic-adrenal system. Then the vasomotor nerves are overly active and the blood vessels of the nose are always more constricted than is normal. If the blood supply to the nose is more constricted because of the effect on the sympathetic-adrenal system, then the reaction to a cold stimulus must be delayed.

If one were to take a normal muscle which had already been stimulated and stimulate it a second time, the response would not be the severe contraction which followed the first stimulus but a delayed reaction with a slow recovery.

When the skin and the vasomotor system are stimulated with cold and constriction of the blood vessels is already present in the nose, the already constricted state cannot be greatly increased. The temperature within the nose falls a little but does not become normal.

Dr. Shurly asked me to repeat what I said about treatment with hot and cold water showers. I have advised my patients to take a warm water bath until they were comfortably warm all over and then follow it, not with tepid water, but with cold water thrown on themselves, first on the legs, thighs, abdomen or chest. If the patient who was not used to such a treatment remained chilled after this brief application of cold he was immediately to take a warm shower until he became adjusted to the cold water and could stand much more of it.

I have advised in every instance in which I have had the opportunity that from childhood the vasomotor system be reeducated by following a tepid bath with a cold sponge bath when the child is very young and later by the use of showers daily.

The myalgias of the neck I have noticed a great deal, and they may have some connection with vasomotor states.

SYMPOSIUM ON BRONCHIECTASIS

Medical Aspects. Dr. Simon Leopold (by invitation), Philadelphia.

Etiology. Dr. H. P. Schenck, Philadelphia.

Roentgenologic Aspects. Dr. Eugene Pendergrass (by invitation), Philadelphia.

Surgical Aspects. Dr. Julian Johnson (by invitation), Philadelphia.

Bronchoscopic Aspects. Dr. Gabriel Tucker, Philadelphia.

These five papers will be published in full in later issues of the Archives.

DISCUSSION ON PAPERS IN SYMPOSIUM

Dr. Frederick M. Law, New York: To my mind, Dr. Pendergrass' paper was the most practical of all the papers presented. It illustrated beautifully the necessity for close cooperation between the bronchoscopist and the roentgenologist. As could be seen from the films, roentgen examination of the lungs for bronchiectasis is not a simple procedure. The laryngologist's cooperation is absolutely necessary to help the roentgenologist supply a roentgenologic diagnosis, considering the present financial state of the private patient. In a clinic the proposition is different, but the laryngologist does not come and ask for a quick examination, dropping some iodized poppyseed oil down the trachea and asking to have the film developed in five minutes and a diagnosis of bronchiectasis made.

Dr. Leopold and other speakers have said that in about 90 per cent of cases bronchiectasis is due to sinus infection. I would say that the percentage is higher.

For instance, in the first case presented by Dr. Tucker the patient was said to have normal sinuses. I was in the back of the room, but I am sure that if the film were properly interpreted, the diagnosis would be chronic infection of the antrum. One should learn to be more careful in the interpretation of detail in the examination, not depending on the opacity of the sinuses but looking at the membrane and the periosteum.

Dr. T. E. Carmody, Denver: I appreciate very much what Dr. Tucker said because I have observed a number of similar cases in which the condition has been cleared up. The patients have not become bronchiectatic. I agree also with Dr. Law, for I have yet to see bronchiectasis without sinus infection.

Dr. Watson and I have done a great deal of work on children. Dr. Leopold spoke of bronchosinusitis. Dr. Watson has suggested this, and we have found that many patients have had sinus trouble early in life. Every child who has reached the age of 1 year and has not had a cold belongs in a museum, because I do not believe that any such child exists. There is always some slight infection in the sinuses, but whether there is a congenital bronchiectasis I do not know.

However, laryngologists who have done a tonsillectomy and then bronchoscopy —Dr. Myerson and Dr. Iglauer have done this often and I have done it occasionally —have almost always found blood, and if blood can be found in the trachea, why is it not possible to find infectious or other material? If a foreign body can be aspirated, why not a clump of bacteria?

With regard to dropping the iodized poppyseed oil into the nose, Dr. Schenck's experiments are very nice and show a great deal. I tried this procedure with my patients who had cleft palate and learned that whether oil was found in the chest depended on the amount put into the nose. I tried putting in just a few drops, and

the first roentgenogram showed nothing. I tried using a larger amount, and then it did show on the roentgenogram. I tried it with children when they were asleep and also when they were crying and would aspirate forcibly, and I did not see any difference in the roentgenograms as to how much they aspirated, provided a sufficient amount was placed in the nose.

I do not believe that the epiglottis has so much to do with getting the oil into the piriform sinus. It is known that secretions frequently get into the piriform sinus, and there is no reason why they cannot spill over into the larynx. They usually do drop into the laryngeal space and, of course, into the bronchi.

I was much interested in Dr. Schenck's saying that clinically he found 80 per cent and roentgenologically 90 per cent of bronchiectasis complicated with sinusitis. I don't know but what I still agree with Dr. Law. Some of the first work, such as that of Robertson on infection from the tonsil by the lymphatic route, was not mentioned by any of the essayists.

Trauma to the pharynx was spoken of. Coughing may cause this, and in children who have whooping cough some trauma at the bronchial tree must occur.

I believe that when there is sinus infection the sinuses should be treated, but I also believe that, as Dr. Tucker advised, one should aspirate the bronchi.

Dr. George M. Coates, Philadelphia: It is hard to discuss this admirable group of papers without some overlapping. I want to emphasize, in the first place, something mentioned by Dr. Leopold and I think by all the other speakers, the value of teamwork, which is even greater in the treatment of bronchietcasis than in the treatment of other diseases. The internist or pediatrician, the roentgenologist and the thoracic surgeon must cooperate, and I do not believe that the rhinologist can be left out, because the association between the sinuses and the chest was definitely proved when Rider and Mullin did their work in 1921. Of course, as much was not known then about bronchiectasis as is known today. Fenton and Larsell did their work in 1936, when a great deal more was known about it.

During the influenza epidemic in 1918, in the base hospital where I was stationed in France there was the same mortality, I suppose, which obtained everywhere, and it was high. In the early part of the epidemic an autopsy was performed on every patient with influenza-pneumonia who died, and all the sinuses were investigated at each autopsy. There was not an autopsy, as I remember, and I saw the vast majority, in which infection was not found in the accessory sinuses, and it was found in a large proportion in the middle ears also.

Every winter a large number of children 3 or 4 years of age are sent to me by the pediatrician with a diagnosis of early or simple bronchiectasis. Examination of the sinuses is requested, and if sinus disease is confirmed, sinus treatment. These children have persistent all winter coughs, which may or may not be productive, with a small degree of fever most evenings. In practically 100 per cent I have found some sinus disease, the presence of which was confirmed by roentgenologic study.

The diagnosis in the case of these little children is not hard because as a rule it is so evident. The disease is usually in the posterior sinuses and often in all the sinuses, with a postnasal drip. With an examination of the posterior part of the pharynx, use of nasopharyngoscope and securing of a history one can make a diagnosis.

Thanks to Dr. Proetz's introduction of his displacement method, incipient bronchiectasis can almost invariably be cleared up. It may recur the following winter but can be cleared up through persistent treatment. If irremediable or irreversible damage has been done, a cavitation granulation develops. Then other methods, for example posture drainage, bronchoscopic treatment or lobectomy, must be used. I think the rhinologist can, for the most part, clear up incipient bronchiectasis in young children by nonsurgical treatment.

Dr. John J. Shea, Memphis, Tenn.: I will review only Dr. Schenck's comment on the cocainization of the pharynx. The act of swallowing protects the larynx in the division of solids from the air. When the pharynx is cocainized the act of swallowing is inhibited.

At the end of each tonsillectomy I keep the child on the table until return of the functions of the superior constrictor muscle, which may easily be ascertained by touch. The physiologic means of controlling the hemorrhage of the adenoids and preventing fluids from going into the larynx is then at hand. If the superior constrictor muscle is functioning, the child will swallow rather than aspirate.

Dr. Robert L. Goodale, Boston: I have been much impressed with two points: first, the emphasis placed on the early, or preventive, stage and, secondly, the comment on the late, or irreversible, stage.

It seems to me that two factors in the early stage are responsible for bronchiectasis. There must be some damage in the bronchus with obstruction or some inflammatory process with infection. Infection alone, in my opinion, does not account for bronchiectasis. In a great many cases of sinusitis bronchiectasis never develops. On the other hand, in a large number of cases of bronchiectasis there is sinusitis. The foreign body or traumatic element seems to me to be almost the determining factor in the development of bronchiectasis. For this reason the early stage, the preventive stage, is the most important one in the life of the patient. The foreign body must be removed. The pediatrician must be impressed with the importance of early examination. After the foreign body has been removed, the patient must be kept under observation, with repeated bronchoscopic or roentgenologic examinations, until all damage has disappeared.

Unfortunately, many patients are seen only after irreparable damage has been done, and in those seen at the Massachusetts General Hospital in the pulmonary clinic, the factor which produced the bronchiectasis in the beginning may no longer be active in the etiologic sense. By that time the patient has the bronchiectatic cavity in the chest and he also has sinusitis.

Conditions suitable for surgical intervention are best treated in that way, and this includes surgical treatment of the sinuses as well as of the chest, and, as Dr. Johnson mentioned, the more complete the operation the more satisfactory the result.

During the early period, bronchoscopy is of great aid and should be an adjuvant to the surgical management of the chest.

One point was not emphasized, but it comes out when one reads the report of 400 cases by Dr. Kenneth M. A. Perry and Dr. Donald S. King.

I had the privilege of working with Dr. Perry and examining the sinuses in those 400 cases. He did not emphasize in his paper, and I should like to comment on, the low incidence of extension of the bronchiectatic process after the bronchiectasis was established.

Of 400 cases, he noted only 7 in which the bronchiectasis developed in another lobe or extended in the same lobe. I mean not just an exacerbation of the process but an extension of it, that is an increase in the cavity.

Dr. George B. Wood, Philadelphia: The one point I want to call attention to is the queer way in which a great many persons use the term "lymphatic infection of the chest from the throat." Lymphatic infection of the chest from the nose and throat is a practical impossibility. I have done a good deal of experimental and anatomic research, and there is no connection between the cervical lymph nodes, per se, and the lymphatic system of the chest except a few peripheral anastomoses between the supraclavicular lymph nodes, which are just above the clavicle, through the skin, and the posterior submental lymph nodes and a few peripheral anastomoses between the peritracheal lymph nodes and the bronchial lymph nodes.

The lymph of the head goes into the deep lymph nodes of the neck, which are divided into two groups, the posterior and the deep. The posterior group drains the nasal sinuses, the nasal pharynx and the posterior part of the pharynx and terminates in the anterior group, and the latter group terminates in the jugular lymph trunk, which on the left side joins the thoracic duct before entering the blood stream and on the right side goes directly into the blood stream at the junction of the subclavian and internal jugular veins.

Experimentally I have tried to induce pulmonary tuberculosis in hogs by infecting the tonsils and hence the cervical lymph nodes. None of the hogs had

pulmonary tuberculosis until they acquired miliary tuberculosis from the breaking down of the last gland in the cervical chain and the tubercle bacillus entered the blood stream. I am convinced that no lymph connection between the nose and throat and the lungs exists.

DR. SAMUEL IGLAUER, Cincinnati: There is a third factor, which is not usually considered, in the relation of the sinuses with the bronchi. It is assumed either that the sinus disease came first and the bronchus became infected later or that the bronchus became infected and, as one speaker tried to show, the nose became infected from the bronchus.

There is, however, a third possibility, and that is that the two became infected at about the same time, and what Dr. Coates has said is in confirmation of this process in the case of acute pneumonia. I think that a similar observation was made in Panama among soldiers who had died of pneumonia. They had both sinusitis and pneumonitis, and apparently the two conditions were acute and synchronous.

The sinusitis may not subside and the bronchial infection may not subside, and then there is the combination, which does not mean that one follows the other.

It has been mentioned that children under ether anesthesia aspirate blood during tonsillectomy. Dr. Myerson has demonstrated this, and it has been shown in Cincinnati that such aspiration occurs in over 60 per cent of children who undergo tonsillectomy. Of course, the patient is under anesthesia. The normal guardian, the reflex of the larynx, is in abeyance, and that is not a normal condition.

It has always been hard for me to believe that when a patient is asleep he aspirates material into his trachea, but probably this does occur more frequently than is known.

Another circumstance which is not usually mentioned, but which has been demonstrated with animals by Bullowa, is that peristalsis occurs in the bronchial tree in addition, of course, to the ciliary action. Whether the peristalsis in man is very active I do not know, but possibly it is present and eliminates foreign material from the bronchial lumen.

Dr. Pendergrass spoke about the method of introducing iodized poppyseed oil for fluoroscopic examination, and I should like to show slides of a simple method which I described a good many years ago but which has not been generally accepted. The method is particularly useful with children, and I think that for the laryngologist who does not do bronchoscopy, as well as for the pediatrician, it is of value.

(Slides were shown.)

This method consists of use of an O'Dwyer tube with a second cannula soldered onto it. The O'Dwyer tube is for breathing, as usual, and the second tube, with a rubber catheter which comes out of the patient's mouth attached, is for the introduction of the oil With children this can be introduced simply by intubation, the fingers being used for guidance, or a little anesthetic can be employed.

The advantage with children is that after one gets the tube in and reassures them, one can take them to the x-ray room without any trouble.

After the tube is inserted, one can inject an anesthetic into the tracheobronchial tree through the second cannula to abolish the cough reflex, because in bronchoscopy one of the worst things which can happen to spoil pictures is for the patient to cough. The tube is allowed to remain in place, and the bronchoscopy is done under fluoroscopic control.

Dr. Leopold brought up the question of why certain parts of the lung become bronchiectatic, and he offered some anatomic explanations. As a matter of fact, the region of the lung behind the heart is particularly vulnerable, and I think that large cavities will often be found there and that the presence of the heart in front of this area interferes with the drainage.

I think all the speakers noticed that this is a region of less resistance, so to speak. There must be some anatomic basis for this fact.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: Roentgen therapy has been advocated. Is this useful or logical?

DR. SIMON LEOPOLD, Philadelphia: Dr. Carmody spoke of some studies by Robertson on lymphatic drainage from the tonsillar region. Dr. George Wood discussed such drainage, but if I understood correctly he denied entirely the lymphatic extension of infection from the upper respiratory passages, including the throat and sinuses, and does not agree with those experimental studies, which have to a certain extent been accepted.

I am not qualified to enter into this controversy except from a purely clinical point of view. My comment is this: I cannot find any other explanation for preponderance of infection on the left side in bronchiectasis unless one assumes that there is infection through the routes which other investigators have demonstrated, through the paranasal sinuses to the chest. On the other hand, clinically I have never seen a patient relieved of bronchial asthma or cured of incipient bronchiectasis by tonsillectomy, from which fact I have always assumed that there is no route of infection from the tonsillar region to the peribronchial lymph nodes and to the lung. I have accepted the upper route of conveyance of infection because to do so is the only way I can explain this curious anatomic distribution of unilateral bronchiectasis in view of the distribution of solitary lung abscesses.

In that connection, I should like to comment on Dr. Iglauer's observation as to the peculiar position of a portion of the lung behind the heart. Were that anatomic explanation correct, then I would expect that lung abscesses also would be two or three times more common on the left side. But they are not. Therefore, I do not think that anatomic explanation applies in bronchiectasis. I should also like to mention, in commenting on Dr. Iglauer's remarks, that I did say specifically that bronchiectasis and sinusitis can occur simultaneously.

Obie's contribution to the study of influenzal pneumonia showed that every patient who came to autopsy had an infection in the paranasal sinuses, and I believe that Dr. Coates made a similar observation from his studies in France during the first World War. So the third possibility, or simultaneous infection, is well known and well recognized and has been repeatedly described.

I also wish to agree entirely with Dr. Goodale in being unable to conceive of bronchiectasis from infection alone, without bringing in the factor of obstruction by a foreign body. However, while this is obvious and important, certainly it occurs in the great minority of cases, because infection is much more common than obstruction by a foreign body.

DR. EUGENE PENDERGRASS, Philadelphia: I have few questions to answer, but Dr. Goodale brought up an interesting point with which I was not familiar, and I am certainly going to look into it: the lack of extension of bronchiectasis after once it has developed. This creates a real question and one that would require considerable study to answer, but it is a very interesting one.

Dr. Iglauer has done a lot of work on almost all subjects connected with the respiratory tract. He raised a question about peristalsis of the bronchus. I did not go into that purposely, because the subject is controversial. No two groups of investigators who have gone into it thoroughly will agree. I think probably the best contribution is that of Charet, who unquestionably was able to show shadows, which he interpreted as peristalsis. His work is convincing.

Dr. Galloway asked about roentgen therapy of bronchiectasis. I have to admit that I am guilty of treating patients with bronchiectasis with roentgen rays. I think a good psychic effect was obtained, but I do not believe that the bronchiectasis was relieved at all unless an acute or subacute infection happened to be present.

It is logical to expect that if there is a superimposed subacute infection the result will be satisfactory. This takes me back to what I think Dr. Goodale said, that there is always an obstructive process in the development of bronchiectasis. This is the thesis on which I have tried to build: There must be something plus infection, and that something, I think, is obstruction caused by thickening of the pleura and other factors which disturb the drainage of the lung.

DR. GABRIEL TUCKER, Philadelphia: Dr. Law has taught me one thing: I will never say anything about films of the sinus again. That film was handed to me and said to be entirely normal, and I took the roentgenologist's word for it. I know nothing about it, however.

There is one point I want to make, that when there is a definite organic disease the lung must be treated and cannot be cured by treatment of the sinuses.

Another point about bronchial obstruction concerns removal of a foreign body through bronchoscopy. By foreign body is meant an endogenous, not necessarily an aspirated, foreign body. A thick body of granulation blocking the lung acts the same no matter where it originated. Whether it is a dry body or thickened secretion or was actually aspirated, its removal has the same effect. Foreign body in that sense includes a great deal and certainly must be considered in the etiology of bronchiectasis, particularly in children. When the secretion is removed, the foreign body is removed. The lung is then open, and there is drainage. Bronchiectasis is thus prevented.

DR. H. P. SCHENCK, Philadelphia: There is only one point I should like to mention, the 100 per cent of sinus disease in association with bronchiectasis.

In 1936 I wrote a paper with Dr. Rikern in which we made that claim, and 100 per cent is a dangerous thing to claim in medicine.

Dr. Tucker revised my opinion, with his cases of obstruction by a foreign body. Another element enters in the figuring of statistics on bronchiectasis, the fact that the bronchiectasis may continue after the sinus infection itself has burned out. I think Dr. Goodale has called attention to this fact in some of his articles. It explains why evidence of active sinus disease is not found in 100 per cent of cases.

Dr. Law would probably be able to tell from the roentgenograms that sinus disease had been there but was no longer active.

CASE REPORTS

Laryngectomy: After-Treatment. (Moving Picture Illustrations) DR. ROBERT E. BUCKLEY, New York.

Granuloma Venereum of the Hypopharynx and Larynx. DR. MERVIN C. MYERSON, New York.

Adenocarcinoma of the Larynx. (Colored Moving Pictures) DR. GORDON B. NEW, Rochester, Minn.

Progress of Mycosis of the Mouth and Throat. (Colored Illustrations) DR. J. J. SHEA, Memphis, Tenn.

Chlorophyll. DR. R. F. RIDPATH, Philadelphia.

Tumor of the Laryngopharynx Removed by Thyrotomy. DR. CHARLES J. IMPERATORI, New York.

Science of Chemotherapy. DR. JOHN A. KOLMER (by invitation), Philadelphia.

A brief historical survey of chemotherapy as one of the most interesting chapters in the history of medicine is given. Also discussed are the mechanism of chemotherapy, animal experimentation and empiricism in the development of chemotherapy, the triumphs of chemotherapy in the treatment of spirochetal diseases and diseases due to protozoa and metazoa, sulfanilamide and its derivatives in the therapy of bacterial diseases and the prophylactic applications of such compounds.

DISCUSSION

DR. T. E. CARMODY, Denver: What does Dr. Kolmer recommend for the nausea and vomiting?

DR. FRANK R. SPENCER, Boulder, Colo.: What can Dr. Kolmer tell about some of the newest of the compounds? Can he tell more about their usefulness?

DR. MERVIN C. MYERSON, New York: For the past three months I have been introducing sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) powder into mastoid wounds before closure. I am convinced that the healing time has been materially lessened.

Recently I observed in a Negress an anaerobic streptococcic infection of the scalp and the tissues of the neck which caused a tremendous slough of the subcutaneous fascial tissues. The structures were opened widely and sulfathiazole introduced, which brought about recovery. Everything possible had been tried, and the patient had been losing ground. I feel therefore that the local application of sulfathiazole in wounds is of definite advantage.

DR. GORDON F. HARKNESS, Davenport, Iowa: I should like to have Dr. Kolmer tell something about the salts, such as the sodium salts which are now being introduced.

DR. CHARLES J. IMPERATORI, New York: Does Dr. Kolmer use alkalis with any of these drugs?

DR. THOMAS C. GALLOWAY, Evanston, Ill.: I should like to comment on the recent reports that colds can be cured by a spray of sodium sulfathiazole solution. I should like to know whether such a spray can be expected to stay in the nose in sufficient concentration to act as a bacteriostat.

DR. BURT R. SHURLY, Detroit: I should like to ask what other drugs should not be used, especially with the sulfur salts.

DR. CHARLES T. PORTER, Boston: I had hoped Dr. Kolmer was going to give some up-to-date information on the after-effects and the deleterious effects of the drugs. He passed those over lightly.

I think one of the greatest difficulties in the use of the drugs is nausea, which he mentioned and about which he told something I have found out in a general way, that keeping a constant level in the blood is probably one of the greatest factors in prevention. A level ranging from 3 to 6 or 7 mg. per hundred cubic centimeters is much more likely to cause nausea than a constant level of 10 or 15 mg., provided it is kept constant.

I was hoping Dr. Kolmer would say something about the delayed reactions and the delayed toxic effects of the drugs. I think these are going to do much to determine the public's willingness to undergo intensive chemotherapy.

Dr. Parran, the chief of the United States Public Health Service, has gotten out a small list of delayed results ranging from permanent hepatic damage to delayed paralysis simulating progressive muscular atrophy, and I know no way of preventing such catastrophes or judging when they are apt to happen.

I have just seen a patient who has had two separate type III pneumococcus infections in less than a year and who now has what I think is a delayed hepatic reaction, or complication, from the use of the drug. The patient is intensely jaundiced without apparent cause, and a thorough study in one of the best medical clinics in the country has not produced a diagnosis.

DR. IRWIN G. SPIESMAN, Maywood, Ill.: Some therapeutic agent for the control of pain, such as acetylsalicylic acid and perhaps acetylsalicylic acid and caffeine in the form of a compound, is necessary. Are these contraindicated when sulfanilamide or a derivative is being used?

DR. BERNARD J. MCMAHON, St. Louis: Does Dr. Kolmer think a person can be sensitized by one of the drugs, and will the discontinuance and resumption of its use or changing to another of the compounds show this? I thought this sensitization was occurring in some of my cases. Perhaps it has been a coincidence.

DR. CLAUDE C. CODY, Houston, Texas: What is the effect of the use of the barbiturates with sulfanilamide and its derivatives? Is it deleterious?

DR. THOMAS C. GALLOWAY, Evanston, Ill.: Does Dr. Kolmer believe that organisms become fast to further doses of the drug?

DR. CHARLES J. IMPERATORI, New York: Dr. Kolmer, would it be better to use whole blood or the serum in transfusion in the treatment of septicemia?

DR. WESTLEY M. HUNT, New York: I should like to ask Dr. Kolmer whether he is willing to express any opinion as to the stability of sodium sulfathiazole in solution.

DR. JAMES A. BABBITT, Philadelphia: Does the intake of this drug affect the p_H of the blood?

DR. JOHN A. KOLMER, Philadelphia: Does Dr. Babbitt mean all the drugs?

DR. JAMES A. BABBITT, Philadelphia: I mean sulfanilamide and its derivatives in general.

DR. JOHN A. KOLMER, Philadelphia: I am afraid that is a question I cannot answer.

DR. E. T. GATEWOOD, Richmond, Va.: I should like to asked about the intravenous and intraspinous use of these drugs.

DR. GEORGE M. COATES, Philadelphia: Will Dr. Kolmer say something about the production of fever in sulfanilamide therapy? Is there any way to tell whether it is a recurrent fever or fever due to the disease?

DR. FRANCIS L. WEILLE, Boston: Dr. Lyons, of the Massachusetts General Hospital, has said that sulfanilamide interferes in some cases with the immunity response of the blood and that sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) does not. Can Dr. Kolmer tell us why?

DR. JOHN A. KOLMER, Philadelphia: There have been, and properly so, several questions which deal with the possible toxic manifestations of sulfanilamide and its derivatives in the human being. In the first place, I may say that these drugs as a group are remarkably low in toxicity, which is fortunate in view of their widespread employment.

Possibly the commonest of the toxic manifestations is dizziness and loss of mental acuity, which many of my own patients have expressed with the appropriate term "wooziness." When these compounds are administered to ambulatory patients, I think it is important to warn them with regard to driving their automobiles of possible danger due to a change in reaction time.

Probably next in frequency are nausea and vomiting, particularly nausea, which is especially likely to occur after use of sulfapyridine. The cause of this nausea and vomiting, curiously enough, is not yet definitely understood. Some investigators believe it to be a state of acidosis.

This brings me to the question of whether administration of the drugs can change the p_H of the blood. I myself am not able to answer that because of the difference in the results reported by different investigators. I believe that these drugs may produce acidosis which may result in nausea and vomiting. For that reason I almost always administer sodium bicarbonate with each dose of sulfanilamide or a derivative administered orally.

On the other hand, there can be no doubt that part of the nausea and vomiting is due to an effect on the vomiting center in the medulla. How this can be prevented, I do not know. I have tried many remedies, but there are none that I turn to with confidence.

In addition to these common manifestations of toxicity, the more serious ones must be kept in mind. Hemolytic anemia is not uncommon, but it need not interfere with the chemotherapy unless it becomes severe, because it is easily controlled with blood transfusions. Severe hemolytic anemia occurs occasionally, but in my experience it is not a frequent complication.

One must also keep in mind that sulfanilamide and its derivatives may depress the bone marrow; that they may be toxic for the bone marrow, leading to leukopenia and particularly to neutropenia.

I might state, in passing, that because of this I regard administration of the barbiturates as inadvisable during sulfanilamide therapy, fearing that the barbiturates may add another factor to the depression of the bone marrow. This neutropenia, or leukopenia, if it reaches 2,000 or 3,000 cells per millimeter of blood is to be looked on as serious because it may result ultimately in what is undoubtedly the most feared of the toxic manifestations, namely agranulocytosis. More than 100 cases of agranulocytosis have been reported in the literature, with a mortality of over 40 per cent.

Another toxic manifestation is drug fever. This, I think, is due to an allergy to the compound. I know from experience that patients occasionally possess a natural allergy to these drugs, so that even the first or second dose may produce considerable fever. But in the majority of patients fever develops after more than a week of treatment.

Such fever is always to be suspected when the patient is clinically well but an unexplained fever persists. Administration of the drug should be stopped, because it is dangerous to ignore drug fever. It has on some occasions been the forerunner of agranulocytosis. When administration of the drug is stopped, if the temperature is due largely or entirely to the drug it will promptly subside. If one is in doubt, it is safe then to give 5 grains (0.32 Gm.). If the patient is allergic there will be a prompt response in the temperature. This is a safe clinical test, and my advice is to stop sulfanilamide therapy if the fever recurs. One may be able to switch to another derivative without the production of fever. This can be determined only by actual trial.

Other toxic manifestations include various dermatoses, some of which may be hemorrhagic. My clinical experience has taught me to have little fear of the cutaneous reactions. I have repeatedly seen the eruption disappear although the chemotherapy was being continued. I do not have as much fear of cutaneous manifestations as of blood changes and drug fever.

There are other toxic manifestations, some of which have been touched on in the discussion.

When sulfapyridine is being administered it is particularly necessary to watch the urine for hematuria, because sulfapyridine, sulfathiazole and sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine) to some extent will be precipitated as crystals in the uriniferous tubules. The deposits are called uroliths, and they may produce some renal irritation and hematuria. If the doses of the drug are then reduced or its administration is stopped, there is, as far as I know, no permanent damage to the kidney.

It is true that sulfanilamide and its derivatives, particularly sulfapyridine, may produce toxic hepatitis with focal necrosis of the liver, characterized clinically by jaundice, and there have been fatalities, although I have not seen any. I have seen temporary jaundice in 3 patients, who promptly recovered when administration of the drug was stopped.

In animals—the mouse, the rat and the rabbit—I have been able to produce such hepatic injuries experimentally, but when one considers that over 2 tons (1,814 Kg.) of sulfanilamide and its derivatives is used in this country every year, one realizes that the incidence of toxic hepatitis must be low.

It may be that the patient referred to by one of the members of the association had become sensitized to the compound, because there is always the suggestion that this grave injury to the liver may be due primarily to an allergic sensitization to the drug.

Sulfanilamide and its derivatives have produced polyneuritis of the peripheral type. I can recollect instances with most of the compounds, although I know of none occurring so far with sulfadiazine. The compound most likely to produce polyneuritis is sulfanilyldimethylsulfanilamide, which is widely used in Germany for the treatment of gonorrhea. I am glad that has not been introduced into this country and hope that it will not be, because it is no better than the other compounds for the treatment of gonococcic infections and it undoubtedly produces the highest incidence of peripheral neuritis.

I may say in passing that a year or two ago some investigators thought that when men were getting large doses of sulfanilamide or a derivative there might be a temporary inhibition of spermatogenesis, but the evidence for this, both clinically and experimentally, has not been convincing.

As to the newer compounds, the compound now called azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate) is the drug which Domagk produced in 1936 and called "soluble prontosil." In this country it is marketed under the name of neoprontosil. Incidentally, I have a high regard for it, particularly in the treatment of hemolytic streptococcus infections. It is sometimes better tolerated than is sulfanilamide.

Sulfapyridine is one of the newer derivatives. I believe it is particularly useful in the treatment of pneumococcic infections, but it is useful also in the treatment of streptococcic, meningococcic and gonococcic infections.

The newer compounds are the thiazole derivatives, suh as sulfathiazole. I believe this is the compound of choice in the treatment of staphylococcic infections, for both local and systemic use. It is highly effective against the pneumococcus, and it is effective to some extent against the streptococcus, but its particular field of clinical application is in the treatment of staphylococcic infections.

Sulfadiazine is one of the newest compounds. It has been in use for only about a year. As far as I know, it has not yet been passed on by the Council on Pharmacy and Chemistry, but it appears to be even better than sulfapyridine. Certainly it is less toxic; certainly it produces less nausea and vomiting, and, as far as pneumococcic pneumonia is concerned, it is at least equal to sulfapyridine in curative activity.

I have recently used it in 2 cases of pneumococcic meningitis of otitic origin. One patient, a child, I now believe will make a final recovery. My first experience with the drug, however, is in these 2 cases.

The latest drug added to the list is sulfaguanidine (sulfanilylguanidine). This is a remarkable drug. In view of the fact that it is soluble in the large intestine but apparently is not readily absorbed, it goes through the portal circulation and is returned short-circuited back into the intestinal canal. It is freely soluble and yet will reach the highest concentration in the large bowel of any of the compounds. It is truly remarkable how it is capable of reducing the number of colon bacilli, particularly in the intestinal contents. Some investigators think it has proved of value in the treatment of bacillary dysentery, but as far as I know its particular field of usefulness is in preparing patients for operations on the large bowel. It is notable for use in connection with resection for malignant disease. Large doses are given for two or three days before operation, the last dose on the morning of operation, and the administration is continued for several days after operation. According to several surgeons of my acquaintance it appears definitely to reduce the incidence of postoperative bacterial infections.

I was glad to hear one of the members of the association refer to the use of sulfathiazole locally in the healing of wounds after mastoidectomy. There has been an impression that sulfanilamide and its derivatives when applied locally to the tissues interfere with healing. I am not a surgeon, but surgeons of my acquaintance have informed me that these compounds may be used locally in wounds without interference with healing and that, if anything, they reduce the degree of infection. One may readily surmise that they promote healing and recovery.

Regarding soluble salts for parenteral administration, I am glad to review them briefly because it is so frequently necessary to resort to parenteral administration when the patient is comatose or cannot tolerate the required dose by oral administration. Sometimes the two forms of administration have to be combined to insure that the patient is securing in each twenty-four hours the total calculated for him.

Azosulfamide is obtained in a 5 per cent solution. It can be given intravenously, although I mention that with reluctance because its intravenous administration is still under experimental study.

Sulfanilamide can always be prepared in an 0.8 per cent solution, so that 1,000 cc. carries 8 Gm., or 120 grains. This solution can be given by hypodermo-

clysis subcutaneously. It can be given intramuscularly. It can be given safely intravenously, and it can also be injected intrathecally.

As to the insoluble compounds, such as sulfapyridine: There is a soluble sodium salt of sulfapyridine which can be given intravenously and intraspinally, and I always give it intraspinally in the treatment of pneumococcic meningitis.

I know nothing about the prevention of the infectious common cold by applying a spray of sulfanilamide or one of the soluble sodium salts to the nasal mucosa. I am convinced that the common cold is due primarily to a filtrable virus. Whether these compounds are destructive for viruses, as far as I know, cannot be stated. It may be that a spray in the treatment of the common cold is useful, not against the virus but against the inevitable secondary bacterial infection which follows the initial infection with the virus. I would not be at all surprised if such a spray was found clinically useful and clinically applicable, but I doubt that any of the compounds have any effect on the primary virus itself.

I have already touched on compatibilities. There is a curious lack of information on this point. There is a widespread tendency to give nothing else when sulfanilamide or a derivative is being given orally. Fortunately there is no need for anything else. I agree that sometimes a pain killer must be given. I never hesitate to give my patients acetylsalicylic acid or sodium salicylate. The only drugs I definitely keep away from are the barbiturates, because I am so afraid they may add further injury to the bone marrow.

If my patient needs sleep, I do not hesitate to give pantopon (a mixture of hydrochlorides of opium alkaloids). It is a favorite of mine for the restlessness of acute illness, particularly septicemia.

There is a long-standing tradition that when sulfanilamide or a derivative is being given no sulfur shall be administered, particularly in the form of magnesium sulfate, the idea being that the cyanosis, which is so common and absolutely without clinical significance, may be due to sulfhemoglobinemia. The English have this idea, but there is no uniformity of opinion on it. However, if one can empty the bowels with an enema or secure proper laxative effects with liquid petrolatum, why take a chance on giving magnesium sulfate? Until the question can be cleared up, it might be proper to defer the administration of any sulfur-containing drug as being possibly incompatible. However, if it is incompatible, its incompatibility is only from the standpoint of its production of cyanosis, and there can be marked cyanosis without the least clinical significance.

The only thing to do is to tell members of the patient's family that this change in his appearance will occur, so that they will not be alarmed when they see it.

I am confident that some persons have a natural allergy to sulfanilamide and its derivatives on occasion, although fortunately it is rare. Allergy is usually acquired. Its commonest manifestation is drug fever. The question was asked whether if it occurs with one of the drugs the therapy may be continued with another compound. My answer is yes, sometimes, but do not persist in giving any one of the drugs if it is producing fever. There is danger of grave neutropenia, which may result in agranulocytosis. Discontinue chemotherapy or try another member of the group.

Is it possible for an organism to become fast to sulfanilamide or a derivative? There is little information on which to base an answer, but I do know of laboratory investigations which have definitely shown that organisms have become fast to sulfapyridine. Whether this is possible with other members of the group, I do not know, but probably it is.

The question has been asked: What is preferred in the treatment of septicemia, transfusion with whole blood or transfusion with serum? My answer, without a moment's hesitation, is whole blood, and I modify that with the statement that bank blood should not be used if it is more than three or four days old, because while bank blood older than this may suffice for maintaining blood volume in internal respiration in cases of surgical hemorrhage, it has been shown that after three or four days there is a marked drop in the phagocytic activities of the leukocytes in the blood, a reduction in complement and a reduction in opsonin.

When the patient has septicemia transfusion is not primarily to furnish him with hemoglobin and erythrocytes. The majority of patients do not need such assistance. What the patient with septicemia does need is immunologic assistance—healthy leukocytes and more opsonin and complements—and these are best supplied by whole blood.

Whether the blood is given by the direct or the citrated method makes no difference. The method to use is the method with which one is most familiar.

In the treatment of surgical shock, I think citrated plasma or serum is preferred, because there is less chance of adding to the hemoconcentration, which is partly responsible for the shock. Serum or plasma is preferred to whole blood as far as the treatment of hemorrhage and particularly of shock is concerned.

I do not know anything about the stability of sodium sulfathiazole in solution. I have been able to use it only within the last few months. All I can say is that when I prepare a solution of sodium sulfathiazole, sulfapyridine or sulfadiazine, I administer it promptly. Whether any changes can occur if it stands, I do not know.

Do sulfanilamide and its derivatives affect the p_H of the blood? I have attempted to answer this by expressing my ignorance. I have not worked with this subject personally. I am confused by the reports in the literature. I believe they may and that they may be responsible for acidosis, but I believe the matter must be investigated still further.

Which of these compounds can be given intraspinally? I know that sulfanilamide can, in an 0.8 per cent solution. I know that a 2 per cent solution of sodium sulfapyridine and a 2 per cent solution of sodium sulfathiazole can. All can be given intrathecally, and this route is frequently indicated, especially in the treatment of pneumococcic meningitis and, sometimes, streptococcic meningitis, both of which come to the attention of laryngologists. In order to get at them as early as possible, use a high concentration in the spinal fluid. Do not use any azosulfamide. It does not pass the choroid barrier in the meninges.

Do sulfanilamide and sulfapyridine interfere with the immunity response? I am glad this question was brought up, but I am unable to give an opinion. I have not investigated it and know nothing to draw on for a final opinion. I should be surprised if the compounds interfered much with the immunologic response in the doses ordinarily given to sick human beings.

It may well be that the compounds in large doses will interfere with the immunologic response in the experimental animal, but if they do I surmise that the dose was sufficient to decrease or depress the bone marrow and the reticuloendothelium distributed elsewhere in the body.

In conclusion, what about home versus hospital treatment with these drugs? In the treatment of severe infections, I think undoubtedly the hospital is preferred. Why? Because frequent determinations for free compound in the blood are needed to guide the dose, which may vary from day to day. Blood counts are frequently needed daily or on every other day. These things can be managed when the patient is at home but usually can be handled better in an institution. I think that patients with severe infections requiring sulfanilamide therapy fare better in a well appointed hospital than in the home and whenever possible should be sent there.

Surgical Treatment of Bilateral Abductor Paralysis. Dr. Brien T. King (by invitation), Seattle.

Slides and motion pictures were shown to illustrate the anatomy and physiology of the larynx, the operation performed on a cadaver, the pathology of the human larynx following bilateral paralysis of the recurrent nerve, the pathology of the larynx of a "roarer" horse with unilateral paralysis of the recurrent nerve and the condition of the vocal cords of patients before and after operation, including the amount of motion restored.

It is the cord not operated on that develops the greater range of motion. The operation is an aseptic approach to the arytenoid cartilage. The cricoarytenoid joint is disarticulated and the arytenoid cartilage displaced outward by a suture

which surrounds the cartilage submucously and is passed through the lateral border of the thyroid cartilage. The displacement of the cartilage separates the vocal cords in most instances sufficiently to produce an adequate airway. The omohyoid muscle is attached to the arytenoid cartilage for the purpose of further opening the cords during the inspiratory efforts.

The results of the operation on 29 patients are reported.

DISCUSSION

Dr. Chevalier L. Jackson, Philadelphia: I have been much interested in this difficult problem for a good many years and I have seen many of the unsuccessful efforts to relieve the patient by other methods.

With regard to anastomosis of the nerves, to which Dr. King referred, I have seen a good many of the unsuccessful efforts for the relief of the difficulty by that means.

Ventriculocordectomy, which Dr. Jackson Sr. suggested a good many years ago, is a procedure which he himself abandoned long ago in favor of tracheotomy with a permanent opening, generally with the valvular cannula. He condemned ventriculocordectomy, both because it was mutilating and sacrificed the voice of the patient, which could so well be preserved by tracheotomy, and because it did not relieve the patient of the use of the tube in the great majority of cases.

I have not tried this operation myself yet, and I think a good many members of the association have been waiting to hear Dr. King's presentation before trying it.

I have seen just 1 patient, the last shown in the motion pictures. I saw her both before and after the operation, and I have seen her several times since. The excellent result in her case is not entirely revealed by the motion pictures, because it is obvious in looking at her larynx that she has an adequate airway.

After operation, as Dr. King said, the cord operated on stays to the side and the function of the voice is taken care of by the residual motility of the cord not operated on. This is the desired result.

Dr. Thomas C. Galloway, Evanston, Ill.: After all the years laryngologists have been struggling with the distressing condition of abductor paralysis, it is rather a miracle that Dr. King has discovered something which is so mechanically sound, so simple and so practical. I think laryngologists owe him a great debt for this operation.

I had treated 5 patients previously with coagulation and an attempt at ventriculocordectomy. In 2 the results were fairly satisfactory. Then I tried Dr. King's operation. I presented this case recently in Chicago. It was interesting because the King operation was done on one side and the more recent Kelley modification of the King operation on the other.

The paralysis had been present for about ten years. I used a local anesthetic; it is not difficult to do this operation with local anesthesia. During the first operation, when I asked the patient, a woman, to breathe deeply, I saw the omohyoid muscle move. After I had attached it to the arytenoid cartilage, it moved the cartilage when she attempted to breathe deeply, and after the operation when she was asked to take a deep breath she was able to pull the arytenoid cartilage laterally very well. The good result lasted for six weeks, and then the previous condition recurred.

I had the privilege of seeing Dr. Kelley's demonstration of a different approach at a meeting of the American Academy of Ophthalmology and Otolaryngology last year. It seemed to me to be a much simpler procedure. It is difficult to cut the inferior constrictor muscle in order to rotate the thyroid cartilage far enough to the side to get good access to the arytenoid cartilage. Dr. Kelley has modified the incision, making an oblique incision over the thyroid alae. A window is made through the thyroid cartilage and the arytenoid cartilage picked up. Dr. Kelley, at least as I understand his operation, dissects out the arytenoid cartilage and then attaches the vocal band to the external perichondrium and the muscle. I modified this operation slightly.

When operating for the second time—on the other side—I cut through the perichondrium with a curet and through the cartilage with a larger curet. The opening is easily enlarged, but it is not so easy to pick up the arytenoid cartilage as one thinks.

It has recently been stated that the arytenoid cartilage may be variable in its position on the cricoid cartilage and that in the female it may be more lateral than in the male.

By inserting a laryngoscope I found it easy to pick up the arytenoid cartilage with a cleft palate hook after I had cut through the internal perichondrium, and then it was easy to dissect it free. It is important, as Dr. King emphasized, that one completely disarticulate the cricoarytenoid articulation. When the operation is finished, only the muscular attachment of the vocal process is left.

It seems wise to preserve the arytenoid cartilage. This gives one a much better guide and traction, as Dr. Seed has emphasized. Dr. Seed, as the result of a careful anatomic study, said that one must make the attachment anterior to the center of rotation of the arytenoid articulation. I partially transfixed the cartilage anteromedially to the center of rotation, through the vocal process and under the mucosa. My patient has an excellent result.

It is important, I think, to recognize the fact that my first operation probably failed because I did not have enough room within the alar cartilage, and one must make the window large enough so that one can pull the arytenoid cartilage through.

As I have said, I performed this operation on only 1 patient. That was eight months ago, and she still has an excellent voice and an adequate airway, which of course she did not have before.

DR. LOUIS H. CLERF, Philadelphia: I am intensely interested in this subject, first because of the need of doing something about bilateral paralysis of the larynx and secondly because I have operated in 6 cases. I am glad to relate that there was only one failure, that is, complete failure to restore motion of the vocal cords, which are still in the median position. The patient is still dependent on her tracheotomy tube. This is no fault of the King operation. Failure was due to a mechanical difficulty which I was not able to overcome. I injured the cartilage to such an extent that it was difficult to attach the omohyoid muscle to it and the latter pulled out.

I have since learned that there is another way of doing the operation, and I am interested in the suture which Dr. King passes around rather than through the arytenoid cartilage. The cutting of the arytaenoideus muscle is a practice, I believe, which he now employs routinely, and it certainly ought to add to the mobility of the arytenoid cartilage.

There is one interesting phase of this operation which I did not appreciate: The lack of movement of the cord operated on was disappointing. Most of the motion was on the side operated on. In studying the motion pictures I saw that most of the movement was on the side not operated on. I am interested to find out why that is. I assume Dr. King can explain.

As to the size of the glottis: When one views the larynx with a mirror, one is not impressed with the size of the glottic chamber, yet the patients are free from the symptoms they previously had, notably dyspnea during sleep. They are able to cork the cannula and decannulate with complete freedom. Thus the size of the glottis is deceiving as one views it with the mirror.

I was interested to learn that the omohyoid muscle had the particular function mentioned. In the last case in which I operated, the patient had had a large thyroid gland and the operation on it was extensive. When I tried to correct the bilateral paralysis, hoping to use the omohyoid muscle, I could not find it. I therefore simply cut the arytaenoideus muscle, mobilized the arytenoid cartilage by cutting well around the capsule, perforated the mucosa and then sutured the arytenoid cartilage to the thyroid cartilage. Incidentally, the result was the best I have secured. The patient is my most recent one, and I do not know whether the good result will be retained.

DR. S. F. KELLEY, New York: I had the pleasure of meeting Dr. King about three months ago, and I corresponded with him after I had done the arytenoidectomy.

If Dr. King had introduced his operation the year before, I do not think the arytenoidectomy operation would have originated.

My interest in this subject was caused by an unusual circumstance. A friend of mine was being sued by a woman who had bilateral abductor paralysis as a result of a thyroid operation. I tried to help him and looked over the literature to find, as I explained in the paper I wrote, an operation which offered a fairly certain chance of success.

I called up several men in New York whom I knew to have performed an operation for this condition. The results in the early cases were not very favorable, because the capsule of the arytenocricoid joint was not cut, that is, the results were delayed. I could not take any chance on this patient's losing her voice to the extent that she could appear before a jury and make a favorable impression, and I had to make it possible for her to get rid of the tracheotomy tube. A consultation was held and the larynx studied.

An old surgical principle, taught to me by my professor of surgery, that nature seldom tolerates a cavity but will fill it with either muscle tissue or scar tissue, was my guiding principle. I thought that if I took out the arytenoid cartilage nature would create enough scar tissue in its place to pull the paralyzed cord over to one side and give the patient a larger airway. At that time I did not know of any one who had had a complete and successful result with the King procedure.

I decided that if I could make a window in the thyroid cartilage and dissect out the arytenoid cartilage the cavity caused by the extraction of the latter would provide a sufficient airway, and that when it filled with scar tissue this would pull the cord over to one side and give the patient a larger breathing space.

With experimentation, I found out where to make the window. My first operation was done with the patient under general anesthesia. I had a bronchoscope tube in place when I did the operation, and it was not difficult to find the cartilage under those conditions, using intratracheal anesthesia.

However, in working in the dissecting room I decided that some fine little hooks, both double and single, would have to be devised for picking up the arytenoid cartilage. These were made, and I used them in 2 cases in which the result was favorable.

I had no idea of modifying Dr. King's operation when this procedure was developed, and my only object was to get an airway. I had not then read Dr. King's paper, but I can see that he has the greatest number of successful operations and good results to his credit. Therefore, I suppose that if one can do Dr. King's operation the arytenoidectomy has no place.

My thought in writing my paper was that one could not lose anything by dissecting out the arytenoid cartilage and seeing whether nature would be kind enough to give the desired amount of space.

There is considerable difference in the distance between the arytenoid cartilage and the inner wall of the thyroid cartilage in different larynxes. In some persons the arytenoid cartilage can be moved readily, with a resultant good airway; in others it cannot.

DR. MERVIN C. MYERSON, New York: I should like to pose this question for Dr. King's consideration: As the thyroid cartilage is closely applied to the arytenoid cartilage in many instances, is it not a fact that one could eliminate the suture of the latter to the former and depend entirely on the tension of the omohyoid muscle and on suture of the arytenoid cartilage to that muscle?

I should also like to ask Dr. King if he would not stress the degree of tension as opposed to the degree of relaxation of the omohyoid muscle in his suturing of the muscle to the arytenoid cartilage.

Also, on the basis of his experience can he tell what degree of airway is consistent with a good voice? I think this is the fundamental consideration in any operation for the elimination of the tracheotomy tube, because, after all, the objective is to give the patient a good voice and a good airway at the same time.

I should also like to ask Dr. King to stress a bit more the matter of placing the suture through the perichondrium, because in doing that I met with one of my failures.

DR. ALLISON T. WANAMAKER, Seattle: It may seem strange that Dr. King has been so much interested in the larynx. I might say that he has been a goiter specialist for a good many years and a very good goiter surgeon.

I was responsible for Dr. King's giving his first paper before the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., at Spokane, Wash., two years ago, and at that meeting he presented his first 3 cases. He was not altogether satisfied with the results, although the patients, especially 2, were markedly improved.

After that meeting, we talked the problem over a good deal. I suggested that he cut the interarytenoid muscle. He went a step further and decided that he ought to free the arytenoid cartilage itself. I saw him do this in the dissecting room. He demonstrated the operation and I am sure that from that time on his results have been much better.

He operated on a patient on whom I had done a tracheotomy, that is the nurse, and I know she has been much improved. She is carrying on her work every day.

I have another patient, a woman, on whom I hope I may get Dr. King to operate. She had severe tracheobronchitis, which resulted in bilateral paralysis. I cannot altogether understand the mechanism which would bring on the paralysis, but she is now wearing a tube and there is a great deal of contraction of scar tissue around the arytenoid cartilage.

DR. GABRIEL TUCKER, Philadelphia: I should like to ask if Dr. King has tried the bilateral operation. If he has not, does he contemplate doing so?

DR. BRIEN T. KING, Seattle: Dr. Galloway brought out some interesting points. My experience with the arytenoid cartilage has been that it accidentally damaged on occasion, and this is what occurred when I attempted to suture through it in my first patient. The cartilage was fractured and the lower half of it sequestrated.

With regard to the 4 patients in whom the arytenoid cartilage was lost or removed, in some of them I have been able to remove the tube and in others not, but they do not have, on the average, an airway equal to that of the patients in whom I did not remove the cartilage but displaced it outward. I feel that the vocal process of the arytenoid cartilage is what pulls the vocal cord outward and holds it there, while if the arytenoid cartilage is removed the vocal process is detached. As I have seen postoperatively, this liberates the cord and it does come outward. Thus, while there is some improvement, the cord does not pull over as far and the patient does not have as good an airway but has a better voice.

In answer to some of the other questions, one must compromise between the size of the airway and the quality of the voice. In 3 cases I have obtained such a big opening that the voice has been poor. In other words, the other cord does not compensate enough to create the sort of vibrations necessary to produce good sound. The best I have been able to figure, in answer to Dr. Myerson's question, is that 4 to 5 mm. is the most desirable space.

I routinely make a laryngoscopic examination when the interarytenoid muscle and the joint capsule of the arytenoid cartilage are cut. In other words, when the cartilage is disarticulated and two sutures are then placed around it at that point, a laryngoscope is passed, and while I pull on the suture I can see the amount of opening secured. If it is not enough, I mobilize the cartilage a little more and then fasten it out.

Another thing, the rotation of the vocal process, is important, and Dr. Galloway touched on it somewhat. If the suture is passed through the vocal process, the process will often be fractured and the objective lost. It would be better, in my opinion, to pass two sutures, one below and one above the vocal process and tie them to the thyroid cartilage. I have been doing the operation that way in the dissecting room. The suturing can be done submucously, just around the perichondrium. I have stopped suturing through the cartilage because of fractures which occur. The cartilage frequently sequestrates.

In one case in which I fractured the cartilage and the lower end sequestrated, I passed a small needle around the fibrous substance of the vocal cord, brought that out and tied it, and this procedure served fairly well. If one has the misfortune to break off or fracture the vocal process, I advise placing a suture in the vocal cord and pulling it out just the same. In my case the cord remained out reasonably well and there was a fairly good airway.

Dr. Tucker asked if I had operated on both sides. I have, but there is not as much motion in either vocal cord as there is in some of the cords I have shown. That is why I stressed the importance of getting a good airway by getting sufficient displacement the first time, because, as every one who has spoken has said, the cord operated on remains more or less fixed. If both are operated on they both remain more or less fixed. There is an airway but the voice is much more husky.

With regard to the occupations of the patients I have operated on, 1 is a school teacher, 1 is a secretary, 1 is a repair man in a telephone exchange in a town of 35,000, 1 is a dancing teacher and 1 is a woodsman, and none has had to change occupation on account of the change in the voice. Most of the patients are housewives. Of the 3 men who have been operated on, 1 is a hardware merchant, and he is still able to make a living.

Paralysis of the Larynx. Dr. Louis H. Clerf, Philadelphia.

It was observed in a study of cases of the common form of laryngeal paralysis, namely abductor, or posticus, paralysis, or that it occurred most often as a complication of operative procedures on the thyroid gland and in cases of tumor not of laryngeal origin. The position of the vocal cord or cords with relation to various etiologic factors and the subsequent changes without apparent restoration of the nerve supply are discussed.

Nodules of the Vocal Cord. Dr. Chevalier L. Jackson, Philadelphia.

True nodules of the vocal cord occur at the junction of the anterior and middle thirds of the cords. They are generally bilateral. They may occur in children as well as in adults. Their cause often seems obscure, and their differential diagnosis from other benign tumors is sometimes difficult. Treatment is by rest and reeducation of the voice and, in many cases, by surgical removal. A series of cases, in most of which the nodules were removed through direct laryngoscopy, are reported and the indications for the method and its technic discussed.

DISCUSSION

Dr. Gabriel Tucker, Philadelphia: The first question is when and when not to remove the nodules.

Dr. Jackson has well emphasized the fact that these nodules usually occur in association with a generalized inflammatory condition of the larynx and that a period of vocal rest before any surgical procedure is carried out is important.

I believe that absolute rest of the voice until the cord is completely healed is the most important thing after proper removal of the nodes.

If the nodules are bilateral it is questionable whether they should be removed together. Nodules of the vocal cord are different from the other lesions in the larynx. With a lesion which involves the anterior commissure or the whole cord, the question of adhesions in the anterior commissure has to be considered and one side should be done at a time. However, if the two nodules are taken off together, of course superficially, the two sides would be helped at the same time.

I was much interested in the next to the last case Dr. Jackson mentioned. I have not had an opportunity to treat a prominent singer for the removal of vocal nodules—and I do not think I want it particularly—but the change in the tone of voice is something which must be carefully considered. I was glad to know that this patient came out all right and did not have any change in her voice. The laryngologist takes a great deal of responsibility in such cases.

Dr. Jackson and I saw another patient in whom a similar lesion existed. It was not a true node of the vocal cord, as the margin of the cord was involved on both sides for some distance, anteriorly and posteriorly to the usual nodule point. We both decided that that patient should have vocal rest, as she was an opera singer. We prescribed vocal rest for three months, and I have some photographs of her larynx, which show that her cords are perfectly normal. Her voice has come back to normal and she is again singing satisfactorily.

I should like to ask one question: Has Dr. Jackson had experience with any of the crooners?

I recently took care of a man who gave voice imitations, and one of the local papers, I noticed, stated that he had been consulting a laryngologist and he might lose his voice. It was a serious matter. Fortunately he was cooperative. He had no trouble after removal of the nodules from both sides. His voice is perfectly good.

Dr. Claude C. Cody, Houston, Texas: Not infrequently one encounters on the Gulf Coast a condition which presents some difficulty in classification, that is hypothyroidism. Just as people from some sections of the country have hyperthyroidism, those from other sections have hypothyroidism. The difference between nodules of the vocal cord and the lesions of the vocal cord associated with hypothyroidism is the difference in the size of the edematous area, bilaterally located at the junction of the anterior and middle thirds.

In the early stages of the condition, when the vocal cords are in adduction the nodes due to hypothyroidism appear and when the cords are in abduction they almost disappear. In the later stages, they become infiltrated instead of edematous and it is with considerable difficulty that they can be distinguished clinically from true nodules of the vocal cord.

So characteristic is the effect of hypothyroidism on the vocal cords in the early stages that a differential diagnosis of hypothyroidism may be made from an examination of the larynx. In the later stages, however, the only differential sign with which I am familiar is the basal metabolism rate, which may or may not be that of hypothyroidism. If it is not, one must resort to the therapeutic test.

In the first two or three weeks, treatment with thyroid substance will correct the condition. Later a cure takes longer, and in some cases in which the infiltration has become fixed it is necessary either to give the voice complete rest or to remove the nodules.

Dr. Charles J. Imperatori, New York: I was glad to hear Dr. Jackson's classification of growths on the vocal cord and his attempt to reclassify them in a workable manner.

I feel that in some instances these nodules are heaven sent, because they stop some singers from thinking they can continue to sing when they have no voice at all.

I have always looked on the condition which Dr. Jackson classified as an organizing hematoma as a hemorrhoid. This is a rather indelicate thing to have in one's larynx, but histologic examination shows that there are a small artery and vein surrounded by hyaline tissue, with the hematin more or less scattered throughout and completely surrounded by a capsule.

My conception of the formation of the organizing hematoma is that in his attempts to sing the patient traumatizes the larynx. There is a break in the epithelium. In the attempt at healing, the capillary bridges which immediately form squeeze on the granulation tissue, as it were, and there is an incipient organizing hematoma.

I feel that in the treatment of nodules of the vocal cord rest is the important thing. Dr. Herbert Curtiss, who had a large practice among singers, expressed the opinion that he could clear up the nodules by vocal rest. I have never seen a nodule of the vocal cord in a singer clear up of itself. I have seen the nodules disappear completely with vocal rest in persons who were not singers.

If there were two vocal nodules, I would agree with Dr. Tucker in taking the two caps off at the same time.

The clinical observation which Dr. Cody has made is edifying, and I will certainly take advantage of it.

I feel that direct laryngoscopy should be the method of procedure in the removal of nodules from the vocal cord. It is a precise method, particularly with Dr. Tucker's improved speculum for visualizing the anterior commissure. Incidentally, an instrument which I have used for a number of years is of help. I call this a subglottic forceps; it has an offset so that one can actually see the tips of the cups grasping the nodule or a larger tumor, such as an organizing hematoma.

Dr. Thomas C. Galloway, Evanston, Ill.: Laryngologists assume that nodes on the vocal cord develop from vocal trauma, but may there not be nutritional or other factors which affect the epithelium and the tone of the underlying tissues? I have thought thyroid extract helped clear up beginning nodes in patients with low basal metabolic rates. Is it not possible that other general factors are important, such as vitamin A deficiency, with its tendency toward hyalinization, and vitamin B deficiency and anemia?

Dr. Ernest M. Seydell, Wichita, Kan.: I should like to refer to the organizing hematoma. I have had 3 patients whom I saw in the early stages, and by merely slitting the hematoma and letting out the blood I prevented the formation of a secondary nodule.

Dr. Chevalier L. Jackson, Philadelphia: In regard to Dr. Tucker's question as to whether I have had any experience with crooners, I have to say no. There was a persistent rumor at one time that a well known crooner was in the Bronchoscopic Clinic, but he was not and I do not know where the report originated. I did hear in a roundabout way that he had been advised to have something removed from a vocal cord and then was told that while removal might make his voice clearer it would spoil the peculiar quality so valuable to him in his crooning, and I believe he never had anything done with it if he had such a lesion.

Dr. Cody's contribution to the discussion was interesting indeed. I have only seen, as I remember, 1 or 2 children with hypothyroidism who had laryngeal changes such as he described, but I am afraid that I have not taken hypothyroidism into consideration at all in treating adult patients with nodules of the vocal cord. I will bear in mind that factor.

Dr. Galloway spoke about the problem of vitamin deficiencies playing a part, and I can only say I have no recollection of any cases in which I found such a factor. The patients have all been robust and in good general health, but I know that this does not always mean that there is not some vitamin deficiency or other nutritional factor involved.

As to the question of removing two nodules at once, as Dr. Tucker and Dr. Imperatori have advised, I think perhaps the objection to working on the two cords at the same time is largely theoretic, and I believe at least half of my patients with bilateral nodules had the two removed together. I follow this procedure particularly when general anesthesia is used. I have not seen any bad effects from removal of the two nodules at the same time.

When one nodule is definitely enlarged and inflamed, it may be better to remove it first and observe the patient a little longer before deciding to take off the other one. I am glad to know that Dr. Tucker and Dr. Imperatori both feel that there is no objection, in most cases, to removing the two nodules at the same time, because I always like to do an operation in one stage if I can.

Dr. Seydell's observation in connection with the treatment of these inflammatory lesions in the early stage is a valuable contribution. I think that such treatment would eliminate the damage to the opposite cord and to the same cord, which certainly does follow the prolonged presence of the tumor. Dr. Tucker's paper of some years ago brought out convincingly the point that serious damage is done to the larynx, both to the cord which has the lesion and to the opposite cord, by the mere presence of the nodule and the strain of phonating while the nodule interferes with the efficiency of the approximation. So the earlier one can get rid of these lesions the better.

If there is a definite subepithelial hemorrhage localized at one place on the cord, it will rarely be absorbed and disappear during a regimen of rest. If the whole larynx is inflamed and congested, I believe it might be better to wait a little while with the patient on vocal rest to see how much improvement will take place, but I think that Dr. Seydell's advice on early treatment is well taken.

I have seen only 1 or 2 patients in whom the lesion was that early. Most patients have the nodules for weeks or months before they report to the laryngologist for treatment.

Papillomas of the Larynx. Dr. E. N. Broyles, Baltimore.

Multiple papillomas in children are serious, requiring frequent removal, tracheotomy and, at times, bronchoscopic removal.

In a series of cases here reported treatment was with an estrogenic substance (amniotin), which seems to have diminished the multiplying and spreading characteristics and made the surgical treatment far more satisfactory.

DISCUSSION

Dr. T. E. Carmody, Denver: I should like to ask Dr. Broyles if he used radium or roentgen rays in any of these cases.

Dr. Frank R. Spencer, Boulder, Colo.: I should like to ask Dr. Broyles if he knows, from his own experience or from the literature, any method which will prevent recurrences. Laryngologists are all annoyed by the recurrence of papillomas of the larynx, especially in children.

I appreciate what he said about estrogen. Are there any other methods of treatment or remedies which offer hope?

Dr. Ernest M. Seydell, Wichita, Kan.: How often is the preparation administered?

Dr. E. N. Broyles, Baltimore: I have tried the treatment of papillomas with roentgen rays in the past. I did not find it satisfactory.

In the use of amniotin, the idea is to spray the larynx in order to change the epithelium from the flat to the adult type, and in the cases which I reported the effect seemed definite. I apply the amniotin by spraying, using an ordinary powder spray with a curved tip.

Dr. Ernest M. Seydell, Wichita, Kan.: How often?

Dr. E. N. Broyles, Baltimore: Once a week.

Physical Rehabilitation of the Laryngectomized Patient. Dr. W. W. Morrison, New York.

This paper will appear in full in a later issue of the Archives.

DISCUSSION

Dr. Robert E. Buckley, New York: I think Dr. Morrison has answered every question I could ask on the after-care of the laryngectomized patient. His paper has been most interesting and instructive.

I had a suggestion not long ago from a physician who was commencing to do laryngectomies which struck me as being valuable. He said, "When a patient requires removal of the larynx, I have one of my assistants start instructing him how to develop an esophageal voice before I take the larynx out." I think that is an excellent idea.

Dr. Henry B. Orton, Newark, N. J.: I have been much interested in the rehabilitation of the voice following laryngectomy.

I cannot agree, however, that the patients seem to have the idea that they are completely cut off from outside life and that they are unhappy. For instance, I can cite a case in which I performed a laryngectomy on an Italian. He could not read or write the English language, but he could speak it. Before he left the hospital he was using the esophageal voice.

I start my patients talking early. I think it is important to send a previously laryngectomized patient in to talk to the patient as soon after the operation as possible, because the newly laryngectomized patient is apt to be depressed for the first two or three days. His friends come in and he tries to talk and realizes that he cannot. Then, if some one who has had the larynx out comes and talks to him it gives him wonderful encouragement.

The artificial larynx which has been discussed is used by all laryngectomized patients but is usually discarded.

Laryngectomized patients, for whom I have had a class for many years, learn to talk much more quickly when instructed in a group, in which they are trying to surpass one another, than when instructed individually.

DR. LOUIS H. CLERF, Philadelphia: Dr. Morrison referred to the suprahyoid and infrahyoid muscles. I should like to ask whether removal of the hyoid bone during laryngectomy materially affects the patient's ability to produce a good pseudovoice.

DR. CHARLES J. IMPERATORI, New York: I should like to add my praise of the fine work Dr. Morrison is doing in New York, particularly in showing how this pseudovoice is produced.

I recall a patient I have seen over a period of ten years who fainted several times while developing the pseudovoice, and I can readily see why he fainted. He undoubtedly produced some disturbance in the mediastinum, in that it was pushed to one side.

Regarding the artificial larynx, all but 1 of my patients have discarded it.

There is another apparatus—and I hope Dr. Morrison will speak about it—which is placed on the outside and which hardly requires any education.

His remarks regarding the care of the patient after laryngectomy are pertinent. I do not think one should do a laryngectomy without having the patient return regularly over a long period so that one can watch for recurrences.

DR. CHEVALIER L. JACKSON, Philadelphia: I heartily agree with everything that Dr. Morrison and the other speakers have said about the superiority of the buccoesophageal voice over the voice produced by the artificial larynx. I think Dr. Morrison has presented the case against the artificial larynx eloquently.

It is the practice at the Bronchoscopic Clinic to see that patients do not start to work with the artificial larynx until they have had at least six months' work on the development of a buccoesophageal voice. After that time, if they have not been successful in developing the voice, by all means, let them use the artificial larynx.

A few years ago I investigated the feelings patients had about their voices after laryngectomy, and I was interested and surprised because the majority of the laryngectomized patients said that their voices were good and the majority of the patients who had undergone laryngofissure said that their voices were only fair.

I had one experience like Dr. Orton's with the Italian. A man from Venezuela who could not speak English before his operation but who started to study it while he was in the hospital made some recordings in both languages when he developed the buccoesophageal voice.

I should like to ask Dr. Morrison what his feeling is about the insurance disability of the laryngectomized patient. I always used to consider that a laryngectomized patient had a total permanent disability, but now that so much evidence is being accumulated on rehabilitation and so many patients are back at their original occupations within a month or two after operation I wonder what stand one should take.

DR. W. W. MORRISON, New York: Dr. Tucker's suggestion about beginning the instruction before operation is an entirely new idea with me, I must confess, and I only wish I had an opportunity to try it.

In answer to Dr. Orton's comment, the importance of an early start is extremely valuable. The mental depression often reaches its peak in the first few postoperative days, and the tremendous help and stimulus given through a visit, and it need last only a few minutes, of a previously laryngectomized patient who produces

an esophageal voice is an enormous psychic aid. Working in a group, with the resultant effort to excel one another, also provides a stimulus and is particularly helpful when one is working with ordinary clinic patients, some of whom are not very intelligent and do not understand what one is trying to do but will try to imitate their fellow-pupils.

As to Dr. Clerf's question about the muscles: Theoretically, if one could leave undisturbed all the muscles associated with the hyoid bone, I think the functional aspect, the matter of producing a pseudovoice, would be easier for the patient, but I must confess that I have seen 2 or 3 patients who learned to produce a usable pseudovoice after having had the most extensive sort of an operation followed by sloughing which destroyed many of the muscles.

With regard to Dr. Imperatori's question, there is a certain element of exhibitionism in some patients. I had one who would bring his tube out and use it often, freely and loudly, and he really enjoyed this aspect of it, but he simply did not try to develop a pseudovoice.

In regard to Dr. Jackson's question, I think most of the insurance companies still agree that total laryngectomy with the wearing of a tracheotomy tube constitutes total disability. The question of rehabilitation undoubtedly enters, and arguments will be raised.

I had one of the most interesting experiences with a Chinese patient who was learning the English language and had his larynx removed. The esophageal voice fits the Chinese well because they talk in short syllables which have a vowel in the center. This patient learned to speak Chinese in a pseudovoice well and can still sing-song. His speech is certainly extremely queer to listen to.

Dr. Imperatori mentioned a new apparatus. I have had no experience with it, but I spoke to Dr. Green, in New York city, about it. It is called the "sonovox" (not yet available commercially) and employs an entirely new principle. It has two oscillating membrane bone conduction receivers applied one to each side of the neck and hypopharynx, and the apparatus makes these vibrate at a given tone, something like the tone of the ordinary voice. This sets the air in the hypopharynx into vibration, and the patient can talk without any effort beyond holding the chest still so that he does not blow. It is an extremely interesting development, about which little is known as yet. I hope that at some future date I shall be able to report on it or, better, have Dr. Green give a report on it.

Mycotic Infections of the Respiratory Tract. Dr. William D. Gill, San Antonio, Texas.

This article will be published in full in a later issue of the Archives.

DISCUSSION

Dr. A. Q. Penta (by invitation), Schenectady, N. Y.: In looking over the excellent program of the society, I was particularly attracted by the title of Dr. Gill's paper, and at the last minute I wrote and received permission from the secretary to take part in the discussion of this most interesting subject. My discussion of Dr. Gill's splendid presentation will be limited for the greater part to the showing of a short moving picture film on the growth of molds. In making this film a motor-driven time photo camera was used. Each second on the screen represents two hours of culture growth.

The titles which introduce the film may seem elementary; nevertheless, it is most important that in the study of mycology one have a basic knowledge of the general nomenclature of the character and structure of fungi. Briefly, the fungi belong to the part of the vegetable kingdom known as thallophytes. They may be parasitic, saprophytic or facultative parasitic. Structurally, they are composed of two distinct parts, a vegetative portion called the thallus or the mycelium and a reproductive portion usually consisting of rounded cells, called spores. In their simplest form fungi multiply by budding or by the formation of hyphae which may intertwine into a filamentous masslike structure, called a mycelium. Fungi are

classified principally according to the manner in which spores have produced their shape and color. As Dr. Gill brought out, for the purpose of clinical study, fungi may be divided into three distinct groups: (1) yeastlike fungi (2) moldlike fungi and (3) higher bacterial forms.

That these various fungi under favorable conditions may produce destructive lesions of the upper respiratory tract was well brought out in the group of lantern slides shown by Dr. Gill. The fungi which I believe should be of particular interest to the otorhinologist are the aspergilli, which are characterized by a basal stem and a stalk with a spore-bearing head. On account of their resemblance to a brush, this species of fungi derive their name from the word aspergillium, meaning a brush which is used in the Catholic church for the sprinkling of holy water. These fungi are widely distributed in nature and comprise one of the most common genuses of molds. In recent years both the allergist and the otorhinologist have come to realize the frequency of sensitivity to aspergilli as a cause of asthma and allergic rhinitis and the failure to cure if the treatment does not include desensitization to these offenders. The role played by these fungi in the causation of bronchial asthma and allergic rhinitis has resulted in the addition of a new chapter on allergy. Today no allergic test is complete unless cutaneous testing for molds is carried out.

Although many species of the aspergillus genus are considered nonpathogenic and some are used to good advantage commercially, they may, under favorable conditions, particularly if the patient shows a sensitivity to the fungus, initiate a localized inflammatory process. In the field of otomycosis it has been definitely established that certain species, in particular Aspergillus niger, which is considered nonpathogenic and is commonly found in the external auditory canal, may under favorable conditions initiate an inflammatory process. In a case recently called to my attention by one of my associates, inspection of the involved ear revealed the external canal and the area about the lower tympanic membrane to be covered by a black moldlike substance after the removal of which small ulcerated areas were visible on the underlying surface. Culture of the material on Sabouraud's medium produced a luxuriant growth from which A. niger and Aspergillus fumigatus fisheri were recovered. The outstanding pathogenic species of aspergilli is A. fumigatus, which has a predilection for the tissues of the bronchopulmonary tract. To the members of this society who are interested in bronchology, I wish to say that mycotic infection of the lungs occurs more often than is generally suspected. In the differential diagnosis of pulmonary disease this condition should always be considered. Every bronchoscopic clinic should routinely have all aspirated specimens of sputum examined for fungi and carry out cultures on Sabouraud's medium. By carrying out this simple procedure in the clinic of which I am a member, my associates and I have been able to establish definitely a diagnosis of pneumomycosis in several cases of pulmonary involvement in which a previous diagnosis of tuberculosis had been made. Regardless of what organs of the body are involved, the final diagnosis of the mycoses rests with the laboratory, which should be employed as soon as possible if proper treatment is to be instituted.

It is fortunate that in potassium iodide, sodium iodide and ethyl iodide there is a group of drugs which if used early will prove efficacious in the treatment of many of the fungous infections. In the treatment of mild infections, ethyl iodide inhalations alone will often bring about beneficial results. Ampules of this drug containing 1 cc. may be obtained, and when an ampule is broken into a handkerchief the drug, because of its tendency to evaporate quickly, must be quickly inhaled. A few years ago I became very much interested in an experiment carried out by a large electrical concern showing the value of refrigeration in the preservation of vegetables. It was noted that when a tomato had begun to be moldy, the process could be completely arrested by placing it in a refrigerator. When the temperature in the refrigerator was allowed to rise the moldy area became larger. Conversely, when the temperature was lowered there was no more progression of the moldy process. From this experiment I became interested in the possibility of refrigeration as an aid in the treatment of the severe types of mycotic infections which would not respond to iodide therapy. The technic to be used would be similar

to the one employed by Dr. Temple Fay in his work on cancer. Dr. Fay and his co-workers have definitely shown that refrigeration, with very low temperatures, will often arrest the growth of cancer tissue. This thought may seem unpractical, and yet in theory it is logical, and perhaps some day workers in the field of mycology may utilize the process of refrigeration in the treatment of progressive mycotic infections which will not respond to any other form of treatment. Dr. Gill has so completely discussed the treatment of the mycoses that I find it impossible to add anything of further importance.

DR. JOHN J. SHEA, Memphis, Tenn.: Allergic persons are more susceptible to mycotic infections than persons who are not allergic, and it is often difficult to get an allergic patient free of a mycotic infection. Desensitization helps. There is no rule on dosage, and one must depend on the local reaction to each dose and not give another injection until the previous reaction has subsided.

(Slides illustrating the lesions caused by Monilia [including thrush], Torula and Aspergillus were shown.)

After operation on the sinuses, especially after radical operations on the antrum, it is not uncommon for the convalescence to be complicated by an acute fungous infection which requires only 1 or 2 irrigations with a 1:4 solution of potassium permanganate to clear it up.

Such infections occur after some radical mastoid operations. When I clean out the cavity at the time of operation I wipe the interior with a paste consisting of bismuth subnitrate 1 part, iodoform 2 parts and paraffin (B. I. P. paste) and then rinse it with alcohol, and the little iodine or iodoform paste that is left will keep down the fungous contamination.

The three drugs I use are iodine, thymol and sodium benzoate. Sodium benzoate is especially valuable in the treatment of the patient who cannot take iodine.

Mycotic infections are more prevalent in the South than they are in the colder climates.

DR. T. E. CARMODY, Denver: Speaking of iodine, I find that when one iodine preparation is not well tolerated another one will be.

I frequently find that if I change from potassium iodide to compound solution of iodine U. S. P. the patient will take it without any trouble. This form can be given in milk, and the gastric irritation will not take place.

I recently treated a patient with actinomycosis with sulfanilamide. I think all the sulfanilamide did was to get rid of the extraneous infection, probably a streptococcic infection, and the iodine and the roentgen therapy used are clearing up the actinomycosis.

Dr. Shea told me about sodium benzoate some time ago, but I have not had occasion to use it. Dr. Shea and Dr. Gill have had more experience with actinomycosis because they come from a moist climate. I do not see this condition frequently, but I do come across a case once in a while. I also see some of the other infections in and around the mouth.

The main help given by the sulfur drugs in all cases of mycotic infection is to get rid of the extraneous infection.

DR. F. R. SPENCER, Boulder, Colo.: Many bacteriologists admit frankly that they do not know as much about mold and fungous growths as they should, although this does not mean that they do not know a great deal. Such papers as Dr. Gill's may help us spur them on to a more extensive study.

In some cases the condition is difficult to diagnose. A year or two ago there was a patient in the Colorado General Hospital who had actinomycosis of the chest, with a fistula in the wall of the chest at about the fifth intercostal space, near the sternum. It was not easy to find Actinomyces in the discharge. The patient was examined bronchoscopically several times. He did not stand the continuous use of any one type of iodide well but did improve by changing from one type of iodide to another, as Dr. Carmody has suggested, and improved enough so that he was finally lost track of. He had been treated and examined by a number of physicians without the actinomycosis having been suspected and without receiving any treatment for fungous growth.

If the Southern members will present papers on fungous growths from time to time, I believe that other members of the association will be more alert. I believe the Southern otologists see the growth of mold in the external auditory canal at least once a day. I am sure I see it only once a week.

DR. SAMUEL IGLAUER, Cincinnati: Dr. Gill's paper is important because one of the greatest mistakes laryngologists make is in overlooking fungous infections, and when such infections are found on culture they are regarded as a contamination from the air.

I recently had a patient with infection due to Bacteroides, and the laboratory technician at first thought it was an accidental contamination, but further study showed this organism in the blood and in the lateral sinus. One should not overlook the possibility of a fungous infection when examining the slides. I think one of the most common mistakes is in overlooking actinomycosis when the patient gets swelling in the neck. A malignant growth, tuberculosis, or possibly, syphilis is considered, but as a rule actinomycosis is ignored.

I mentioned Bacteroides, which actually does not belong among the organisms under discussion, because it is pleomorphic and therefore difficult to identify.

It has been sugested that fungous infections occur particularly in hot climates. As a matter of fact there is more actinomycosis in Minnesota, and other parts of the Middle West than there is in the South. I do not know whether the supposition that heat and moisture have so much to do with this infection is correct.

Dr. Gill referred to a case of blastomycosis of the frontal sinus which I reported some years ago. The patient was a soldier who had been overseas during World War I. He had a swelling over his frontal sinus, and when he blew his nose the mucosa would protrude from the opening in the frontal bone, creating a pneumatocele, so to speak. He has lesions in the spine and in the foot and, I think, also in the mediastinum. The diagnosis was made, as I recall it, from the lesion on the foot. The patient subsequently died, and all the lesions were examined at autopsy.

In the treatment of actinomycosis potassium iodide acts well sometimes, and at other times, despite large doses, it does not. I think when one looks at the pus from the wound one should look for the yellow granules which are so characteristic of actinomycosis.

DR. HOWARD C. BALLENGER, Winnetka, Ill.: I should like to know if there is any value in the local application of iodides, particularly to the ear.

DR. WILLIAM D. GILL, San Antonio, Texas: I was pleased to hear Dr. Iglauer call attention to the fact that there is no geographic area which is free from infections due to molds.

When one searches the literature and begins to correlate the data one finds that reports come from all over the world. It is true that in tropical and subtropical zones the incidence is greater, but regardless of where one practices medicine, one will encounter these infections.

I think susceptibility is induced largely by a change in the body chemistry, which furnishes a suitable substratum on which the mold can grow. It is perhaps an overalkaline state of the secretions which permits the growth. I feel that at least a part of the treatment should comprise attention to the diet, particularly in the withholding of carbohydrates as much as possible.

My experience has been a little contrary to that of Dr. Shea. I find that most of the patients are extremely tolerant to iodides. The little girl who had the large abscess, whose picture I showed, was given 150 grains (9.7 Gm.) of potassium iodide by mouth and 31 grains (2 Gm.) of sodium iodide intravenously every day for some time, with no harmful effect.

The important thing in making the diagnosis when one is dealing with an intractable lesion is to insist that the bacteriologist hold the cultures as long as three weeks. He throws them away in three days if they are not positive; or if he does not throw them away and there is a growth of mold he usually considers

it a contamination and reports the culture negative. The physician thus gets a negative report on the culture when, as a matter of fact, it may not be negative at all.

After all, it is the primary duty of the physician to free as many patients as possible from debilitating conditions. In many instances the proper care will save a life. Many patients with fungous infections which have been treated as tuberculosis finally die, when they could readily have been cured and rehabilitated by the use of iodides.

I did not say anything about otomycosis because it is not properly included under my title, but I found the most effective treatment to be the use of cresatin-Sulzberger (metacresylacetate). It is applied on cotton pledgets, placed in the canal and allowed to remain for twenty-four hours. Then it is removed and wiped away with a good deal of the epithelium. This drug is a bactericide and analgesic, and it is also fungicidal.

I once published an article on otomycosis and described the use of a special fungicide composed of 1 per cent thymol, 1 per cent tricresol and 1 per cent iodine in boric acid powder.

If one cannot see a patient every day, one should use the powder, applying it with an ordinary powder insufflator.

News and Comment

THE EYE AND EAR RESEARCH FUND, INC.

The Eye and Ear Research Fund, Inc., announces a refresher course in the fundamentals (anatomy and pathology) of otorhinolaryngology, which is intended for candidates for the American Board of Otolaryngology. The course will be given at the Illinois Eye and Ear Infirmary and at the Research and Educational Hospital, Chicago, on Wednesday, October 8, from 8 a. m. to 5 p. m., and on October 9, 10, 11 and 12 during evening hours. Applications and requests for particulars should be sent to Dr. Robert Henner, the Illinois Eye and Ear Infirmary, 904 West Adams Street, Chicago.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14-8th Ave., Brooklyn.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. T. E. Carmody, 227-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western.—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of socicties are requested to furnish the information necessary to keep this list up to date.